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The Beattie Smith Lectures.[1]

(UNIVERSITY OF MELBOURNE.)

THE PROBLEM OF NEUROSYPHILIS AND ITS TREATMENT.

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LECTURE II.

IN the previous lecture an endeavour was made to present the outstanding points in regard to the ætiology, pathology and psychopathology of neurosyphilis, and the unity-in-variety concept of this condition was stressed.

To refer briefly again to this lecture, it was shown that insufficient arsenical treatment tended to favour the development of neurosyphilis, but that arguments both for and against a special neurotropic strain of spirochæte remained inconclusive. Figures were shown which revealed pregnancy as a protective agent against neurosyphilis, although in what manner this is brought about is not yet known. The work of Brown and Pearce on tissue vulnerability was referred to, as also the work of Jahnel and the later researches of Robert Dieterle, illustrating the three varieties of spirochætal distribution in the cortex. This was illustrated by a number of lantern slides from Dieterle's work. The significance of the physical constitution was discussed, the pyknic and asthenic types reacting differently in neurosyphilis, and finally an attempt was made to interpret in psychopathological terms the mental reactions of a paretic.

Now it is proposed to take up the problem of treatment and to discuss the newer therapeutic measures recently brought to our aid in attacking the more pernicious and malignant forms of neurosyphilis.

It would appear that the *Spirochæta pallida* may attack any part of the central nervous system, although some portions are infected much more frequently than others. The practical applications of specific therapy show that success is achieved far more easily and more often when neurosyphilis exists in the meningeal and meningo-vascular types and also in those cases of "asymptomatic" neurosyphilis where the only evidence of infection is seen in the cerebro-spinal fluid abnormalities. Early forms of the tabetic type, characterized pathologically by a localized meningitis of the posterior nerve roots and before ganglionic degeneration and involvement of the posterior columns have commenced, yield in a large percentage of cases to routine antispecific treatment. But when tabes has advanced into its ataxic stage, it is as resistant to such treatment as paretic neurosyphilis itself.

Many theories have been advanced to explain why the modern chemical spirochæticides have so little effect in these conditions; but in the absence of positive proof we must rest content with such tentative hypotheses as deficient lipoid solubility of the drug or the fact that arterial disease and perivascular infiltration which accompany the lesion also prevent the spirochæticide from reaching the causative organisms.

Practically the whole prognostic element in neurosyphilis depends upon the amount of cellular damage done at the time of treatment. If the cells are merely incapacitated, as Kolmer says, by the pressure of lymphocytes and plasma cells, the absorption of these may result in partial or complete restoration of function, so that some clinical improvement may follow specific treatment in paresis, tabes and tabo-paresis; but if the brain cells are actually destroyed by the toxins of the spirochæte, treatment may arrest the disease process, but complete restoration of function is impossible.

While the general attitude of the profession seems to be nihilistic towards the results of specific treatment in paretic and tabetic neurosyphilis, there are some who either from innate optimism or clinical inexperience are prone to expect too much from specific therapy. Despite the alchemists, there is no magic in mercury; nor is "Salvarsan" supernatural in its action. It may destroy spirochætes; it cannot regenerate brain cells. The metaphorical sow's ear of neurosyphilis cannot be metamorphosed into the silk purse of perfect health. In early asymptomatic neural involvement complete cures are apparently secured in a goodly percentage of cases, but in symptomatic cases of longer standing the real and usually the only hope is in checking the progress of the infection, with relief from some of the more distressing symptoms and the improvement of functional capacity for the usual allotted span of life (Kolmer[5]).

From what has been said it will be obvious that routine treatment with mercury and some arsenical derivative is not efficient and that syphilis of the central nervous system requires a particular form of treatment. The problem which presents itself here, is merely how best to destroy the spirochætes present in the central nervous system. But the nervous tissue is walled off, as it were, from the general body structures and this leads to a relative impermeability to drugs placed in the general circulation. Theoretically and practically it seems advisable to place the medicament as near as possible to the site of pathological change, utilizing the lumbar subarachnoid space, the region of the *cisterna magna* and the ventricles, as conditions indicate.

Intraspinal Therapy.

The first attempts in the field of intraspinal therapy were made by Marinesco in 1911, followed independently and almost simultaneously by Swift and Ellis. Since then a veritable wilderness of literature depicting different methods and variations in technique has sprung up in America and on the Continent. Much of this is of a controversial

[1] Delivered on October 29 and November 5, 1928.

character, the perusal of which tends more to confuse than clarify; so that a mere reference to salient points will be made here.

Swift and Ellis devised their method of "Salvarsanizing" serum *in vivo* by injecting "Salvarsan" intravenously and drawing off 40 to 50 cubic centimetres of blood about an hour later from which the serum was secured the following day, and injected into the lumbar subarachnoid space after an equal quantity of cerebro-spinal fluid had been withdrawn. Ogilvie modified this method by adding a known quantity of "Salvarsan" to the serum before introducing it intrathecally.

Although encouraging results had been obtained by the Swift-Ellis method, investigators proceeded to work in the direction of obtaining some means of increasing the penetration of the tissues of the central nervous system during antispecific medication. Among these Gilpin and Earley[17] in 1916 advocated spinal drainage on the grounds that a reduction of spinal fluid pressure resulted in hyperæmia and increased production of fluid. Another and later method was that of Corbus, O'Connor, Lincoln and Gardner[18] who sought to bring about the same result in another way. They administered one hundred cubic centimetres of a 15% saline solution which immediately produced a drop in the cerebro-spinal fluid pressure. Some hours later, at a time when it was calculated that this pressure was being restored, 0·6 or 0·9 gramme of "Novarsenobillon" was injected intravenously. And these authors claimed that in twenty-six out of twenty-eight cases of neurosyphilis the procedure was followed by an increase in the arsenic in the cerebro-spinal fluid.

An ingenious method of double puncture has been devised by Gennerich[19] and used by him since 1921. This consists of inserting two lumbar puncture needles a couple of interspaces apart to which are attached sterile containers. After ten cubic centimetres of fluid are drawn off for serological tests, about twenty cubic centimetres are run off into the upper container which is clamped. The remainder of the cerebro-spinal fluid is then drained into the lower container which is then also clamped. To the twenty cubic centimetres in the upper container from 0·5 to 5·0 milligrammes of freshly prepared "Neosalvarsan" are added and, when mixed, this is slowly run back into the spinal canal; the needle is then withdrawn and by raising the lower container the remainder of the fluid is allowed to flow back, thus allegedly washing the "Salvarsanized" fluid up towards the base of the brain.

Gennerich claims good results from this method; but other workers have found it dangerous owing to the development of symptoms of cord irritation and concomitant bladder troubles. At Mont Park we found the method easy to carry out; but while the patient did not show any untoward symptoms such as the irritation referred to, no clinical benefit was manifested. Nevertheless the method seemed to us rational and worthy of a more extended trial.

In their clinical work Swift and Ellis found that 60% to 80% of patients suffering from various types of neurosyphilis whom they treated, obtained a complete clinical and serological arrest; some of their patients have been under observation for ten years without a relapse. Ogilvie, using his modification of their method, claimed a complete clinical remission in 34% of the paretics he treated. Keidel and Moore[20] have reported that 60% of their patients treated by the Swift-Ellis method showed good clinical results. Schaller and Mehrteens[21] noted clinical and serological arrest in 64% of neurosyphilitics, while Stokes and Shaffer[22] four years ago reported on 405 patients with neurosyphilis under observation for from three to seven years and claimed a return to symptomatic and serological normality in 90% of patients with meningeal neurosyphilis, in 74·5% of patients with cerebro-spinal neurosyphilis, in 48·3% of patients with tabetic neurosyphilis and in 38·9% of patients with vascular neurosyphilis. Solomon, of the Boston Psychopathic Hospital, in 1924 stated as his opinion that the Swift-Ellis method of intraspinal therapy is the most satisfactory and is indicated in many cases of neurosyphilis which have not otherwise been successfully treated (Fordyce).

Although the majority of workers in this field has used the lumbar route, a certain amount of experimental work has also been carried out on the intracisternal and intraventricular routes.

In 1914 Berriet injected "Salvarsanized" serum through the sphenoidal fissure into the large basal subarachnoid cistern and recently McClusker, Ebaugh and others have advocated injections of "Salvarsanized" serum into the *cisterna magna* by the Ayer's technique. It is stated by those enthusiastic about this method and among them is Sir James Purves Stewart, that by this means the serum is brought nearer to the seat of cerebral syphilis, that it is just as safe and free of reactions and more practical than the intraventricular route. In fact, the latter method has fallen into disfavour, as it has been found to be without benefit in the treatment of paretics. In a long and penetrating review recently published of the intraventricular treatment by Cestan, Riser and Peres,[23] these authors come to the melancholy conclusion that:

> Although the injection of salvarsan into the cerebral ventricles brings on a happy modification of the humoral syndrome, they have never observed any recovery in the right sense of the word. Never does the dementia undergo a favourable modification; and the action of such treatment upon the clinical evolution of the disease seems very doubtful.

Of all the intraspinal therapeutic methods only the more important of which have been mentioned here, the Swift-Ellis procedure seems to be the method of choice and Fordyce (*loco citato*) gives the following as indications for such treatment:

> 1. All cases of neurosyphilis with definite findings in the cerebro-spinal fluid which are not responding to treatment intravenously, and in which advanced degenerative changes in the lower cord are not present, call for spinal therapy.

2. Cases which have become intolerant to salvarsan intravenously, as evidenced by exfoliative dermatitis, jaundice *et cetera*.

3. In optic atrophy where the initial process is presumably due to a basilar meningitis it is possible to arrest the progress of the atrophy by continued intraspinal treatment.

And the following he cited as contraindications:

1. Neurosyphilis with a negative cerebro-spinal fluid.
2. Cases which are being favourably influenced by intravenous treatment.
3. Markedly alcoholic subjects.
4. The aged with sclerotic changes.
5. Low tabes with advanced degeneration.
6. Cases which do not tolerate the treatment as evidenced by severe headache, meningism or where symptoms of irritation of the lower cord have developed.

The severer critics of the Swift-Ellis technique hold that the beneficial results are brought about by the initial injection of "Salvarsan." But whether arsenic injected intravenously is capable of penetrating cerebral tissues is a very debatable point still and probably no subject concerning the distribution and fate of substances injected intravenously in the treatment of neurosyphilis has excited as much discussion in the literature. While the exact origin of the cerebro-spinal fluid is as yet unknown, it is likely that under physiological conditions this fluid is a form of secretion of the chorioid plexus and ependymal cells which effectively prevent the passage of most drugs into the cerebro-spinal fluid. Hexamethylenamin, uranin and chloroform may pass through and especially the former; but from the standpoint of drugs employed in the treatment of neurosyphilis it may be stated that mercury has never been found in the cerebro-spinal fluid, except when it was injected directly into the subarachnoid space and Catton and Mehrteens failed to find iodine after intravenous injection of iodides.

The literature dealing with the penetration of arsenic into the cerebro-spinal fluid and the central nervous system is somewhat vague, some authorities believing that arsenic does not penetrate, while others claim that it does with great difficulty. Experiments which were carried out last year in the pathological laboratory at Mont Park, using the very sensitive Sanger-Black method with nascent hydrogen to detect small quantities of arsenic, failed to reveal even a trace of arsenic in the cerebro-spinal fluid thirty, sixty or one hundred and twenty minutes after intravenous injection of "Novarsenobillon" or "Tryparsamide." But by first injecting thirty cubic centimetres of inactivated normal horse serum into the subarachnoid space, thereby setting up an aseptic meningitis, arsenic in measurable quantities was discovered in the cerebro-spinal fluid following an intravenous injection of 0·45 gramme of "Novarsenobillon." Several patients who showed an immunity to malaria, were treated by this method and the results so far have been encouraging.

The mechanism of subarachnoid therapy, therefore, may be summarized as follows:

1. The non-specific effects due to subarachnoid and meningeal irritation following an injection of plain or medicated serum.

2. The increased penetration of the tissues by spirochæticides in the blood, as the result of a disturbance of the chorioid-meningeal complex, due to spinal drainage and intraspinal injections.

3. The antispecific activity of medicaments in the serum.

4. Possibly the influence of antibody formation when autoserum is used, as in the Swift-Ellis method (Kolmer).

"Tryparsamide."

It will have been noticed that in this very rapid and woefully incomplete survey of the chemotherapy of neurosyphilis no reference has been made to the action of "Tryparsamide," as owing to its recent rise to prominence in the treatment of paretic neurosyphilis, it was thought advisable to consider it separately.

"Tryparsamide" is a pentavalent arsenical compound of much lower toxicity to animal cells than the trivalent "Neosalvarsan" and it can therefore be used in larger doses. Introduced originally as a specific for African trypanosomiasis this drug was subsequently found to possess penetrative powers above the other arsenicals and hence it became a valuable weapon in the therapeutic arsenal of neurosyphilis. It was used widely in America, where investigators found that its chief danger lay in administrating it to patients presenting optic atrophy.

In this respect the studies of Woods and Moore[24] of 241 patients receiving more than three thousand injections of "Tryparsamide" show that:

Dimness of vision was observed in 10·2% and constriction of the visual fields for form, without scotomas, and with or without diminution in visual acuity, in 5·5% of cases.

In their experiences they found:

1. That 94% of all reactions occurred early in the course of treatment.

2. That visual disturbances bear no direct relationship within certain limits to the dose of the drug administered.

3. That diseases of the central nervous system, especially paretic and tabetic neurosyphilis, are to some extent a predisposing factor to visual disturbances from "Tryparsamide."

4. That preexisting syphilitic disease of the optic nerve or retina is not necessarily a contraindication to the use of "Tryparsamide," but that

5. Before "Tryparsamide" therapy is begun every patient should be examined by a competent ophthalmologist as to visual acuity, fundi and visual fields. And the occurrence of any visual disturbance must form the occasion of a second examination.

The emphasis which these authors lay upon the visual disturbances, may seem a little overdone. At Mont Park last year in the treatment of neurosyphilitic patients I have given over three hundred

intravenous injections of "Tryparsamide" and have only twice noticed any visual disturbance follow the injection. The ocular fundi of patients were examined prior to treatment and those showing signs of optic atrophy were rejected. With the others "Tryparsamide" has proved a very safe drug and I am inclined to agree with Lillie[25] who says that changes in vision, fundi and perimetric fields are probably due rather to syphilis than to "Tryparsamide."

The latest of many reports in the last few years on the beneficial effects of "Tryparsamide" comes from Jaenicke and Forman[26] and was published only a few months ago. These authors conclude that beneficial results of the treatment occur in indirect ratio to the duration of symptoms before treatment is initiated. Improvement is first noted in from the sixth week to the third month after treatment; clinical and serological cures were produced in 5% and clinical and mental improvement in 38% and the best results were obtained in paretics presenting the manic type of reaction.

My own experience with "Tryparsamide" used alone in the treatment of paresis extends to four patients only, in all of whom the psychotic symptoms and physical signs were well marked. Three of these patients have been discharged from hospital; two of them are now back at work. But in order to get the maximum results from treatment, it has been the practice at Mont Park to follow malarial therapy with "Tryparsamide" which, as will be shown later, appears to give the most encouraging results; for apart from its antispecific action, "Tryparsamide" is of great value for its roborant and nutritional properties. But while all who have used "Tryparsamide," are agreed as to its usefulness, it is too early to express a final opinion and wise to hold firmly to the mast of scepticism in the rising winds of therapeutic enthusiasm, since no one can assume at present that the patients are actually cured in the sense of complete extirpation of the disease.

Non-specific Therapy.

From considerations of the ætiological equation in an individual into whose body the spirochætes have gained entry, it would appear that the ultimate fate of the patient is dependent on the individual and constitutional factors which influence the "predilective soil" which the organism invades and in which it finally lodges. The fact that some neurosyphilic infections progress to a rapid and fatal termination while others may achieve a remission and remain stationary for years, leads one to believe that the natural bodily defence mechanisms play a large part in the course of the disease and instances of remissions without antispecific treatment can be explained only by the fact that the body was capable of displaying a maximal reaction to the virus and that the surprising curative results were achieved by the highly developed natural protective powers of the body. Much attention in psychiatric circles is now being bestowed upon the mobilization of the non-specific curative factors in syphilitic therapy.

Our knowledge of the curative defence mechanisms of the body is as yet insufficient to enable us to understand how any form of non-specific protein therapy can bring about clinical improvement in neurosyphilis. And so we are compelled to invoke a theory of vascular change associated with hyperæmia, enzyme action or capillary permeability to account for the benefit which we produce empirically, and then to administer one or other of the known spirochæticides in an attempt to eradicate more thoroughly the ætiological virus.

It would be both tiresome and unprofitable to refer to the numerous foreign protein substances which have been used experimentally in the treatment of paretic neurosyphilis. The two most outstanding, however, are boiled milk and brewer's yeast. Kyrle in 1920 gave intramuscular injections of boiled milk as an aid to specific therapy and in 1925 Urechia and Mihalescu[27] published results of injections of an emulsion of brewer's yeast in the treatment of one hundred and four paretic patients. They claim that of these thirty-five were subsequently more or less able to return to work, thirty-five were improved and thirty-four remained unimproved.

This shock protein therapy seems to me merely a mechanical method of calling forth a certain tissue response, as mechanical as driving a tack into the carpet and just as a tack may be driven home with a hammer, the head of an axe, the back of a spanner or a rolling pin, so may the tissue response be called forth by brewer's yeast, boiled milk or, if you wish, egg albumin.

Many clinicians in the past had noted the apparently accidental benefit of malaria, typhoid, erysipelas *et cetera* upon the course of paresis and the idea subsequently gained ground that the production of an acute fever by other agents might be beneficial in the treatment of tabes and paresis, since no one at that time had had the audacity to produce infection deliberately for this purpose, as Wagner-Jauregg[28] finally did in 1917 by inoculating his paretics with the parasites of benign tertian malaria.

Induced Malaria.

For the last ten years non-specific therapy by the induction of malaria has gradually gained ground, until at present it occupies the forefront of the therapeutic approach to the more malignant forms of neurosyphilis. Many thousands of neurosyphilitics have been treated with malaria and much research work has been carried out with reference to the mode of action of malaria; so that it will be possible for me to give only a very brief outline of this method.

Great care must be exercised in the selection of the strain of malarial plasmodium to be used, since infection with subtertian varieties has proved fatal. Whereas it has been shown that the experimental asexual tertian injections are much easier to cure

with quinine than natural infections resulting from anopheles transmission and while a few investigators have reported remissions, no particular difficulties have been encountered in the treatment of such.

The technique of the inoculation is simple. The malarial blood withdrawn from a patient at the height of the febrile paroxysm may be injected intravenously, intramuscularly or hypodermically. The intramuscular injection is, however, the method of choice. I made over one hundred such injections last year with scarcely a failure. Four to ten cubic centimetres of blood are withdrawn and immediately injected deeply into the muscles at the angle of the scapula. As it is only with the greatest difficulty that the malarial parasites can be cultivated in the laboratory, the strain is maintained by successive inoculations from one patient to another.

As a general rule there is an incubation period of from eight to sixteen days when the inoculation is made by the intramuscular route. The longest incubation period I have noted is thirty-one days and the shortest three days, the length of the period depending possibly on the relative number of parasites in the injected blood. About 5% of patients do not "take" on the first inoculation and must be reinjected. A few seem to possess an immunity to malaria.

The type of fever may be tertian or quotidian; the former is less severe on the patient and the latter type, developing in an elderly or debilitated patient, may necessitate premature termination of the fever. Most investigators have allowed their patients to complete from eight to fifteen febrile paroxysms. At Mont Park we aim at twelve, the number depending, of course, on the state of the patient's general health and how he is reacting to the fever.

It is advisable to make daily examinations of the blood in order to count the number of parasites present. A safe limit, according to Nicol, of Horton, should not exceed thirty-five parasites in twenty-five consecutive fields, a number 2 eyepiece and an oil immersion lens being used for the estimation. When the number of parasites becomes greater, one must consider the advisability of terminating the infection by quinine.

Most success has been observed in paresis and especially in early cases, where the lesion is manifest chiefly by irritative symptoms; but even in advanced maniacal patients remissions have been produced. The treatment has also been used in other types of neurosyphilis and especially in *tabes dorsalis* and some clinicians in Germany and Austria have been so impressed with the success and safety of malarial therapy, that they are using it in early asymptomatic cases as a prophylactic measure.

My own feeling is that malarial therapy is of definite value in all cases of syphilis involving the central nervous system as a means of provoking the natural tissue defence mechanisms and that it should be used in such cases as a preliminary to antispecific treatment either by mercury and iodine or intraspinal injections of "Salvarsanized" serum.

There exist, however, certain contraindications to its use. Patients presenting marked cardiac lesions or pulmonary tuberculosis should not be given malaria and those with coexistent renal trouble should be regarded as "poor risks."

Induced malaria must always remain a specialized form of treatment and on account of the dangerous conditions which may develope and the risk to life which is involved, should never be applied haphazardly or by those who are unable to give the closest attention to the patient.

The following, if they arise during the course of treatment, should be regarded as danger signals, calling for immediate cessation of treatment: (i) The development of any acute illness, (ii) œdema of the limbs, (iii) congestion of the lungs, (iv) persistent vomiting, (v) cardiac failure, (vi) congestive seizures, (vii) tendency to collapse, (viii) repeated hyperpyrexia (that is over 41·1° C. or 106° F.), (ix) pulse rate exceeding 150 beats per minute, (x) respiration rate of or exceeding 60, (xi) pulse pressure falling below twenty millimetres of mercury, (xii) hæmoglobinuria or albuminuria (other than transient), (xiii) anything more than a slight icterus, (xiv) a parasite count of more than thirty-five in twenty-five consecutive fields, (xv) sudden marked increase in the number of parasites, (xvi) failure of the temperature to fall below normal in the interparoxysmal period, (xvii) abnormal somnolence.

Despite this long list of danger signals, malaria is in reality a very safe form of treatment when properly applied, given two things: a pure strain of benign tertian parasites and a competent nursing staff. Under these conditions the risk to life is very small. In the earlier days the malarial mortality used to be set down at 15%. In the last few years it has come down to 3%. In my own series of ninety-six patients treated at Mont Park last year there were no deaths due to malaria, largely, I confess, owing to the unermitting attention and enthusiasm of the nurses in charge of the patients.

The mechanism of malarial therapy is still unknown. It was originally thought to be due to the high temperature being inimical to spirochætal growth; but while this cannot be the sole factor, it has been shown that a greater percentage of good results is obtained when the mean temperature of the paroxysms is over 39·4° C. (103° F.). Malaria provokes a lymphocytic reaction and Bruetsch and Bahr[29] have recently published their histological studies on the mechanism of inoculation malaria. These authors conclude:

1. That the treatment of paresis with inoculation malaria produces during the febrile paroxysms histopathological changes in the brain consisting of proliferating phenomenon of the capillary endothelium which must be regarded as part of the reaction of the reticulo-endothelial system.

2. The absence of perivascular infiltration in the greater part of the cortex in a patient who died at the

height of a paroxysm towards the end of the malarial course, suggests the disappearance of the infiltrating cells during the acute malaria.

3. During malaria and particularly at the time of the febrile attacks plasma cells probably immigrate into the brain vessels taking part in the phagocytosis of the liberated plasmodia.

4. After the retrogression of the perivascular infiltration normal conditions are reestablished in the perivascular lymph spaces resulting in a partial recovery of the ectodermal tissue.

Results of Malariotherapy.

The results of treatment by malaria are very encouraging. The figures obtained at Mont Park, covering a number of unselected neurosyphilitics, are in agreement with those published in the literature and show an almost complete clinical remission in 33% of cases and pronounced general improvement in both the physical and mental state in a further 35%. Of the remainder, a few have become worse, a few have died subsequently and the rest appear to have remained stationary. The patients with early tabes in the preataxic stage appear to do very well, but those in the late stages do not for obvious reasons derive much benefit.

The clinical results generally have been more striking than the effect upon the response to the Wassermann test and spinal fluid changes, although these may undergo favourable changes at the same time. Scherber and Albrecht have shown that the best results are secured by combining malaria with treatment by "Salvarsan" or its derivatives and mercury; at least the serological results are better. These results are also in agreement with the findings of others, including Gerstmann and Wagner-Jauregg himself. The practice pursued at Mont Park is to follow malarial therapy after several weeks' convalescence with weekly injections of three grammes of "Tryparsamide" for eight weeks; then the patient has a month's rest and another course of eight weekly injections of "Tryparsamide" is given, after which the patient reports every three months for observation. Intraspinal therapy, either by the Swift-Ellis or the Gennerich method, is used only for those patients who have a natural immunity to malaria or who appear to have derived no benefit from fever therapy.[1]

One of the earliest patients whom I treated at Sunbury in 1925, was a paper cutter. He was discharged from hospital and has remained well ever since. Another patient is a bank manager who is at present negotiating financial transactions in his bank as well as he did before the onset of his trouble. This particular man who on admission was euphoric, tremulous and slightly ataxic, can now play six sets of tennis without any trouble. Another patient, a bricklayer's labourer, is at present at work on the Shrine. There is another in the railway workshops at Newport who supplements his income by playing in a jazz orchestra at night. Another man is now working in an iron foundry. Another is a carpenter engaged by the Footscray Council. Another tabo-paretic whom I showed some time ago at a clinical meeting and who may be remembered by some of you as the man who had such pronounced delusions about making money on the racecourse, has now joined his wife in a boarding house establishment. One of my lady patients whose husband died just prior to her admission to Mont Park, has married again and says she has never felt in better health. Another lady whose only remaining signs are stiffness and inequality of the pupils, is behind the bar in a well known city hotel. Another of our patients has gone back to his former position in the railway offices and yet another is working in the repair shops of a large city motor garage. This list could easily be lengthened, but enough has been said to indicate how gratifying the results may be from malaria, for without some such treatment, as you well know, practically all these people would now be among those demented wretches into the twilight of whose minds ideas have ceased to cast their shadow.[1]

Other Types of Fever Therapy.

Considering the results which have been obtained by malaria, it is not surprising that investigations have been carried out with other fevers and Plaut, for instance, in 1926 reported his experiences of over six years with the spirochæte of African relapsing fever. Judging from all data, Plaut considers that the results of malaria and relapsing fever treatment are about equally balanced. Malaria has the advantage that it can be suddenly terminated by quinine and that it is possible subsequently to reinfect the patient. Relapsing fever is said to be less dangerous and the treatment easier to carry out, especially as infectious material is easily available by keeping the disease going in mice.

In the same year as the work of Plaut was published, Solomon, Berk, Theiler and Clay wrote on ratbite fever, advocating it on the grounds that the organism can be maintained in laboratory animals and thus is always available for use and that the disease is less debilitating than malaria and that it can be given to patients who are more or less immune to malaria. The infection is easily controlled by "Salvarsan." These authors have inoculated only a few patients, but abundant fever was produced and so far the results are promising (Bassoe). But up to the present time neither relapsing fever nor rat bite fever has achieved the popularity of malaria.

It seems almost redundant to state in a final review of neurosyphilis that subsequent involvement of the highly specialized nervous tissues is best prevented by prompt and adequate treatment of the primary lesion and by examination of the spinal fluid in all patients presenting a positive blood response to the Wassermann test. Haphazard and

[1] At this stage a number of lantern slides was shown depicting beneficial changes in handwriting *et cetera* after malaria and also a number of slides showing the physical state of paretics and juvenile paretics following treatment.

[1] A cinematograph film was then shown of the whole course of treatment as carried out at Mont Park, including pictures of patients before and after treatment. Two patients who before treatment were unable to walk, were shown and pictured again after treatment, walking up and down the ward.

inefficient treatment of any syphilitic lesion is in many cases worse than no treatment at all. Neurosyphilis, even when developed to the stage of evident physical disease, may be checked and in some cases the disease may be completely eradicated; but even so, like tuberculosis, syphilis always leaves scars in it wake. The best results of treatment by the modern chemotherapeutic methods are still obtained when the main damage is confined to interstitial tissues, as in the meningeal and vascular varieties and in early tabetic types before an ascending degeneration of the posterior columns has been initiated. Advanced neural degenerations are, of course, beyond all treatment, but in the majority of cases, though the pessimism of past experiences may darken the prognosis, attempts should always be made by whatever therapeutic agent seems most suitable to check the inevitable progress of the disease. And I agree with Solomon who believes that with the means we now have in hand the majority of the patients with syphilis of the central nervous system, whether the so-called cerebro-spinal syphilis, *tabes dorsalis*, general paralysis or other forms, are entitled to treatment and that, if this is done thoroughly, intensively and systematically, the results will be gratifying.

We sometimes talk airily about curing diseases; but in a great many cases complete cure is impossible. Cure in the literal sense implies the complete eradication of the organisms producing the disease as well as the complete restoration of damaged tissues and impaired or lost functions. In neurosyphilis this is hardly ever possible; but one is frequently able to bring about what may be called a "clinical" cure in which the patient is cured in the sense that the disease is arrested, the causative organisms are destroyed, inflammatory exudates are absorbed and impaired function in the psychic sphere is partially compensated.

The whole position has been stated aphoristically by Harrison who rightly says that "we can put out the fire, but we cannot rebuild the house." Let us then put out the fire at the first signal, before irreparable damage is done, attacking the problem prophylactically by examining the cerebro-spinal fluid of every patient with primary and secondary syphilis and in such patients as those presenting abnormalities, proceed to correct them by appropriate treatment before discharging the patient as cured. Only when such a course is followed rigorously, will neurosyphilis in its protean forms cease to plague mankind.

Coda.

Syphilis has about its beginnings the elements of romance: the very name was born of Jeronimo Frascatoro in a poem about an Italian shepherd lad. Its advent in Europe was dramatic, connected with Columbus and the new world across the Atlantic. Each century that has passed has been as a fresh act in this absorbing play of the pathos and ingenuity of man and in which the *Spirochæta pallida* is starred. Certain of the characters have become classic; certain of their sayings have been embodied in the great realm of medical truth. Kings and queens have trod the boards along with paupers and harlots in this medical "passion play." Madness has crazed the minds of many and still the actors have their exits and entrances. The great curtain of time has already descended upon four acts and when it arose at the beginning of this century, the fifth act commenced with a series of brilliant diagnostic and therapeutic triumphs. It may be that we are now approaching the final *dénouement*. But we have three-quarters of a century before us yet in which with all the powers placed at our disposal by scientific medical research, with practical therapeutics and by hygiene in association with social means we must work to this end, namely, that this fifth act shall also be the last act, as it is in the case of true drama.

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THE MEDICAL ASPECTS OF ACCIDENT INSURANCE AND WORKMEN'S COMPENSATION.[1]

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IN opening this discussion upon the medical aspects of accident insurance and workmen's compensation, I do not expect to tell you anything that is unknown to you. My object is rather to present some of the experiences that I have met with both in hospital and private practice. I do this with two objects, first, that it may promote discussion among those present and the recounting of their experiences may be of help to myself, and secondly, that my own experiences may be of help to other members.

When a patient consults a medical man for treatment of a condition resulting from accident in which he alone is responsible, the only duty of the practitioner is to alleviate and cure the condition. If, however, the accident is the result of injury caused to the patient by someone else or the accident has occurred while the patient was working for an employer, an additional factor is immediately introduced. This factor is one of compensation that may be claimed by the injured person at some later date. It is wise on the part of the medical man if he realize this and is methodical in recording the condition while it is fresh in his memory. In this connexion I would emphasize some points that may be of use to him in the future if he is called upon to assess the disability from the medical aspect.

The first point I would make is that the general condition of the patient should be noted with special regard to the appearance and demeanour and whenever possible the body weight should be recorded. The first medical man in attendance may not be called upon in the event of litigation or arbitration to assess damage. But in the event of litigation or what may be considered exorbitant demands for compensation at a very much later date, possibly six to eighteen months may have elapsed and the notes taken in the first instance, when compared with those at a later examination, will be found to be of very considerable assistance in arriving at a just estimate in this matter.

The second point I would bring under your notice is in regard to head injuries. I have made it a rule in my wards at the hospital that all patients with head injury should as soon as possible have their vision tested with test type and recorded in their notes and their hearing roughly tested with a watch and the distance at which this can be heard recorded. The advantage of this has been brought under my notice on more than one occasion.

In one instance a man who had suffered a severe head injury with fracture of the vault of the skull, made a recovery and passed out of my care. After about twelve months I was asked to examine him by a firm of solicitors. He was claiming a thousand pounds damages from the owner of the motor car which had knocked him down, on account of his injuries, suffering and expenses and the fact that he was as the result of his injury blind in one eye. On examining his notes, I found no record of his vision and I was unable to say that his blindness was not due to his injury and an ophthalmic surgeon who examined him at this time stated that he had optic atrophy and which he considered might have been caused by his accident. He received very substantial damages and it was quite by accident some years afterwards that I heard that he had been blind in that eye for some years before the accident.

Even if no such glaring case be considered, it must be obvious that a record of the vision and hearing, taken as soon as possible after a head injury, must be of value in arriving at a conclusion if loss of weight or hearing is claimed at a much later date. The same necessity and advantages are derived from a record of the reflexes, pupils, knee jerks *et cetera*.

Another point I would make here is that great care should be exercised in discussing the injuries received in the hearing of the patient. In this regard it is to be remembered that, as is the case with those who are ill, it is not always advisable for the patient to know the full extent of his injury. This is especially so when medical terms are used with which the patient is not wholly or even partly acquainted.

I have seen men who have been told that their leg or arm is not fractured, but only broken, make a much speedier recovery and return to their occupation with a much happier frame of mind than they would have if the limb had been fractured. This warning is needed in all cases, but especially in hospital practice where the majority comes under the *Workmen's Compensation Act*.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on March 28, 1929.

Such an injury as a very slight fracture of a vertebra of the spine may have extraordinarily little influence on the man's earning capacity or comfort, but once let him know that he has such an injury, he will ascribe all the aches and pains he may have from a dozen different causes to this injury and not be well for very many months without compensation having been received for a disability that is largely in his mind. If the fracture or the compression is going to cause grave nerve or other trouble, it will do so without his knowing of the injury and he should then be compensated adequately. Two patients under my care illustrate this point.

A man severely injured in a tram accident, arrived in hospital only partly conscious with a dislocated shoulder, five ribs were badly fractured and he had a large effusion of blood in his pleural cavity. He was very dangerously ill for many weeks, but ultimately recovered, but many months later he sought the advice of a physician for pain in his arm. An X ray photograph was taken of his cervical vertebræ and a partial fracture dislocation was found, quite healed, at a point that corresponded with the roots from which the affected nerve originated. Unfortunately the injury was discussed with students in his hearing and the "broken neck" demonstrated with the aid of the X ray film. He was placed in a fixation apparatus and although it is some years now since this happened, he has, I understand, done no work and cannot ride in a motor car on a bitumen road because his broken neck causes such pain at the site of the injury and in his head. He did not complain of this for months and not at all until he discovered the injury.

Another man, admitted under my care in an unconscious condition, had an X ray examination of his skull which disclosed no fracture. He recovered from his concussion and left the hospital. Some months later he consulted me for pain in his arm. An X ray film showed a fracture dislocation of the fifth cervical vertebra quite soundly healed and with not much callus. He was not informed of the injury and was treated with massage and electric treatment for his neuritis which gradually cleared up and he returned to his work and so far as I know, though several years have elapsed, is still following it. He would not have done so in my opinion had he known he had a broken neck.

I would emphasize the necessity for X ray examination in all cases of injury in which bone may have been affected without giving signs that can be elicited by an ordinary manual examination. Often a small fracture without displacement is discovered when none is suspected, but one great value of the examination is the normal finding which can always be produced as evidence and aids in convalescence.

In referring to X ray examination, I would mention one class of case in particular and that is injury to the back, whether caused by falling at work, being run over or struck by a falling object.

No one can see pain and no one but the patient feels it, often there is very little external bruising and it is essential that an early X ray examination be made in these circumstances. They are some of the most difficult to assess. The following is an experience that I have had on more than one occasion, when a patient with an injury to the back has been referred to me for an opinion many months afterwards.

The claimant states that he has pain at the site of the injury, cannot walk or bend with comfort and cannot work. X ray examination reveals no displacement or evidence of fracture, but it often reveals rheumatoid arthritis of the spine in men over forty years of age who have lived laborious lives. Is this condition due to the injury or was it present at the time the injury was received? If an X ray film were available showing its presence at the time of the accident, it is obvious that it is of less moment from a compensation point of view than if it can be proved that it was not present and followed upon the accident. While on this subject, I would urge great caution on the specialists who do X ray examinations, in conveying their opinions on the result of their films without having the full clinical history to aid them in arriving at an accurate diagnosis.

Films shown by Dr. Betts at our last clinical meeting, of spinal injuries held by some to be fracture and by Dr. Betts to be congenital deformities, illustrate one difficulty.

The subject of traumatic neurasthenia, words dear to the counsel for the claimant, the judge and the jury and abhorred by the counsel for the defence, is one which bristles with difficulty. We as medical men meet very genuine examples of this, but we also meet many that are not genuine and to describe which a better term might well be "delayed compensation complex." The first is a real suffering and very distressing to the patient, but in all genuine cases yields to time and efficient treatment, just as the profound depression of physical weakness after a prolonged illness disappears with returning health and suitable environment. The second remains stationary or becomes worse until compensation is received when in many instances it fades like the morning mist. It is difficult for any of us to be sure which is which and much harder for counsel and the courts to agree. It is a term that it is not wise to use in a medical certificate unless the medical man is very sure of the genuine nature of the condition.

The question of straight-out malingering is one that I could speak about for a long while, and of which I could give many instances. The ingenuity of this type of person and the cleverness with which he evades detection are known to us all. There is no royal recipe for detecting it. Careful examination, obtaining their confidence and distracting their attention are the means that sometimes enable you to prove the malingering and these with intuition and experience upon the part of the medical examiner are the only means of saving a miscarriage of compensation.

The increasing number of persons injured by accident treated at public hospitals cannot fail to strike anyone who has much experience of work there. A great many of these are insured against accident and, being carried there in the first place, stay there until they are convalescent. Many of these could pay for private hospital and treatment,

ILLUSTRATIONS TO THE ARTICLE ON THE SHOULDER JOINT BY DR. H. FLECKER.

FIGURE I.

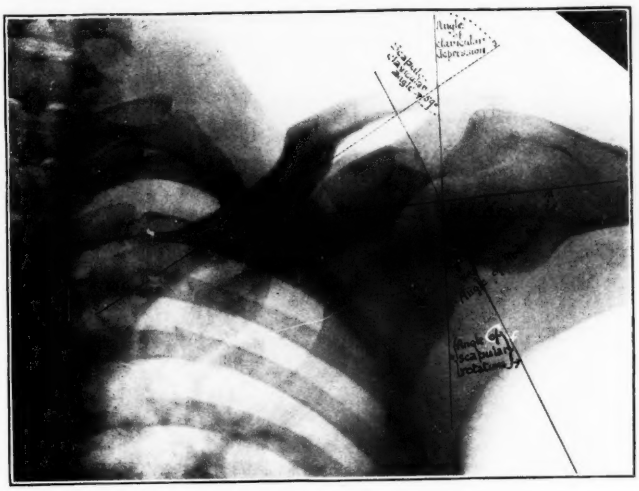

FIGURE II.

Note the relative positions of axes of humerus, scapula and clavicle in different positions of the shoulder joint. The figures represent angles of abduction, scapular rotation and clavicular depression. Accepted anatomical teaching that the humerus does not rotate in the glenoid cavity beyond the horizontal position is manifestly erroneous.

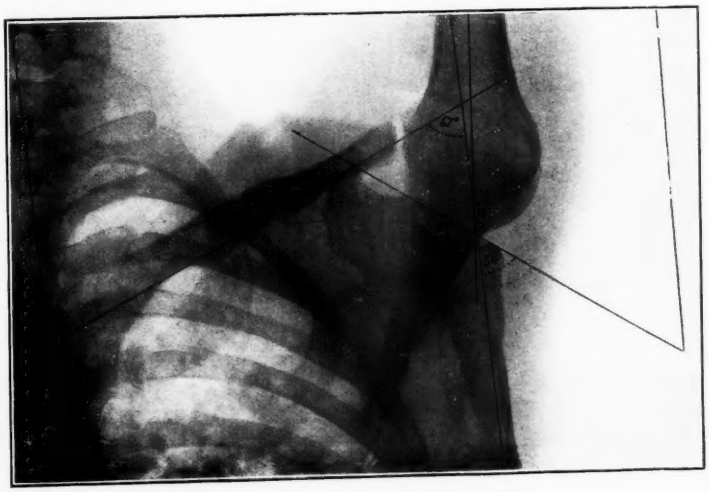

FIGURE III.

ILLUSTRATION TO THE ARTICLE ON THE OCULAR BULB BY DR. H. FLECKER.

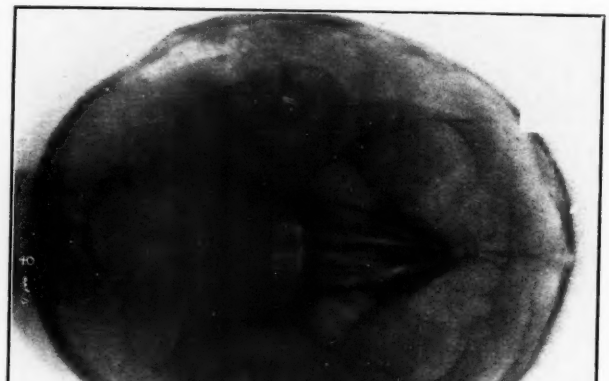

FIGURE I.
Note the contours of several coats of optic bulb on both sides.

ILLUSTRATIONS TO THE ARTICLE BY DR. D. R. W. COWAN AND DR. L. O. BETTS.

FIGURE II.
Lumbo-Sacral Spine, Antero-Posterior View, Showing Defect in Laminæ of Fourth Lumbar Vertebra.

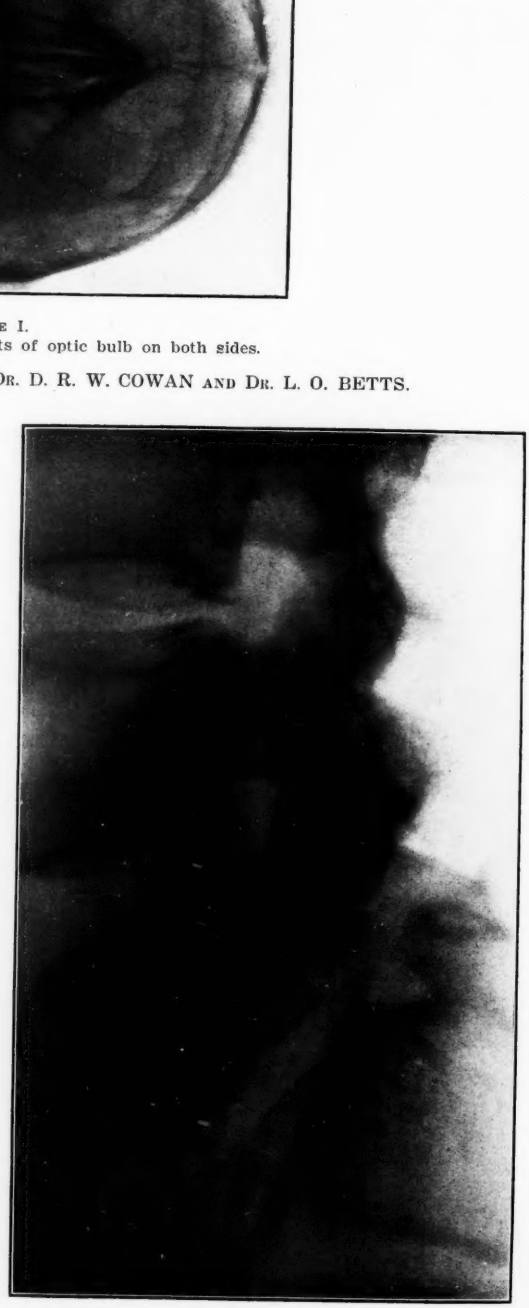

FIGURE III.
Lateral View of Lumbo-Sacral Spine in Patient with Spondylolisthesis.

the principal purpose, one would think, for which the insurance is originally effected. They receive all their treatment in a public hospital without fee to the honorary staff. This is a point which might very well be considered more definitely in this Branch, as the number of cases is steadily mounting. In the chronic convalescent variety, there is no doubt it would be of advantage, not only to medical practitioners, but, I think, to insurance companies also, if this type of patient were treated by a private practitioner who, if paid by the company, would have a definite personal interest in expediting the return of the patient to his occupation.

These are some general aspects of the *Workmen's Compensation Act* that seem to me to call for attention on the part of medical men. There is no doubt that the knowledge that after an injury received while employed the injured person will receive half wages and if permanently disabled full compensation, is a great comfort to a working man with a family. There is, however, a certain risk of some loss of character in that the individual labourer is certainly less likely to attempt to make provision for such an emergency, knowing that in the event of such an accident he will receive treatment at a public hospital and part wages and compensation. If he is a decent man, he is only too glad to get back to his job once his health is restored. But some, and they are by no means rare, who are drawing half wages and are possibly receiving one pound per week from a lodge and one pound per week from a union in certain other cases, are quite content to "enjoy" ill health while they can collect without working, as much, if not more, than their wages. These are very hard to persuade that attendance at an out-patient department is no longer necessary and work is a good thing. I feel that a medical man can help to stiffen their resolve to get well and often by judicious advice help these men to help themselves.

In conclusion I would ask you to treat this paper as one intended to open a discussion on points of interest to all practising medical men and if it elicits other men's experiences and views, I shall feel that it has not entirely failed in its object.

THE MEDICAL ASPECT OF WORKMEN'S INJURIES.[1]

By S. H. SKIPPER, LL.B.,
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APART from statute the employer, broadly speaking, was only liable for injuries sustained by his workmen through the personal negligence of the employer and if those injuries were contributed to by the negligence of the workman or his fellow workmen, the employer could escape liability. The *Employers' Liability Act* extended this liability, but the claim to damages was still based on negligence. The *Workmen's Compensation Acts* gave the workman a remedy apart from the negligence either of himself, his employer, or his fellow workmen and in effect made compensation for industrial injuries a charge upon industry.

The administration of workmen's compensation lies in the hands of insurance companies, medical men and solicitors and in the last resort the courts. The vast majority of claims is settled on the reports of medical men and, therefore, it is of the utmost importance that they should thoroughly understand their duties and responsibilities.

The Workmen's Compensation Acts.

A personal injury by accident arising out of and in the course of a workman's employment and causing him incapacity to earn in some suitable employment the same average weekly wages as he was earning prior to the accident gives the workman a right to claim compensation from his employer. The question whether the accident arose out of and in the course of the workman's employment may be left to the decision of lawyers. The matters upon which medical opinion is sought are:

1. Was the injury due to an accident?
2. Does such injury give rise to an incapacity for work?
3. Is such incapacity total or partial, temporary or permanent?
4. What is or will be the wage earning capacity of the workman in some suitable employment?

Definition of "Accident."

An accident has been authoritatively defined as an unlooked for mishap or an untoward event which is not expected or designed. In most cases it is easy to predicate that an injury is the result of an accident, but in many cases it is extremely difficult to do so.

I take the following case from my own experience.

A man while wearing a felt hat was struck in the frontal region of the skull by a small piece of soft super the size of a pigeon's egg. He complained that he had been so struck, but exhibited no signs of pain or distress and continued his work. A week-end intervened, after which he did another day's work; then followed the Christmas holidays and on the reopening of the works he worked another seven days without complaint, except on the last day, when he complained of a bad headache, which he ascribed—and this would not be evidence in a court of law—to the blow on the head. He eventually died between two and three weeks later of an abscess on the brain in the region where he was struck.

The question of whether or not the abscess was caused by the blow on the head, although ultimately it may have to be determined by the courts, is largely a question of medical opinion.

Medical men, as naturally they must be, are often influenced in their opinions by statements made to them by the patient, but in giving evidence they should be careful to differentiate between what is evidence admissible in legal proceedings and what is inadmissible. A statement made by a workman

[1] Read at a meeting of the South Australian Branch of the British Medical Association on March 28, 1929.

as to the cause of his injury is inadmissible. A statement made by a workman as to his symptoms is admissible. If, therefore, a medical man is required to support his opinion in court, he should be careful to eliminate from the facts upon which that opinion is based anything which is not strictly evidence. For instance, suppose a workman comes to a medical man and says: "I strained myself turning the handle of a sausage machine," this would not be evidence, but if the workman said: "I felt a sudden pain at my work," this would be evidence. The act which the workman was performing at the moment when he felt the strain would have to be proved by what is called direct evidence as distinguished from mere statement made by the workman some time after the alleged accident.

In coming to an opinion, medical men should regard themselves as acting in a judicial capacity and reach their conclusions by a process of careful reasoning after they have made themselves familiar with as many facts as they possibly can.

It was laid down in the case of Wicks *v.* Dowell and Company (1905), 2 K.B., 225, that an employer took his workman with all his disabilities. Two cases occur to me which will exemplify this rule of law.

A man was injured in a bicycle accident, was admitted to hospital and finally discharged with an intimation that he was tuberculous. Some months later he slipped and fell while yarding sheep and injured his back. He now has a tuberculous spine.

In another case a driver had fallen from a trolley and injured his shoulder. The injury was not serious, but he eventually found his way into a mental hospital with general paralysis of the insane patient. The result of the Wassermann test was positive, so the exciting cause of the general paralysis was apparent. At first the medical man consulted reported that the accident had nothing to do with the workman's then condition, but after an explanation of the principle of Wicks *v.* Dowell he expressed the opinion that his condition was due one-third to the accident and two-thirds to his syphilitic history.

In these cases it would be fairly obvious to the legal mind that compensation was payable. It is the old story of lighting up a dormant disease and if the accident does that, then the employer must accept liability as though the accident were the sole cause of the disability.

One naturally turns now to death claims, where the same principles apply.

In one case which I litigated, the workman got a splinter in his finger, which caused it to fester. The poison spread and brought on a feverish condition and in due course pleurisy set in. This was followed by congestion of the lungs and liver which lit up the chronic nephritis, from which the workman had been suffering for years. He eventually died. Death was the result of the nephritis, as the poisoned finger, the pleurisy and the congestion of the lungs and liver had all cleared up. In the examination of his medical attendant the following question was put and allowed by the court: "But for the accident, would death have happened as and when it did?" The answer being "no," the workman's dependants succeeded in their claim.

Here the chain of causation was complete, but it may not always be complete. It may be broken by a *novus actus interveniens* within the principle laid down in Dunham *v.* Clare (1902), 2 K.B., 292, in which Collins, M.R., said:

The question whether death resulted from the injury resolves itself into an inquiry into the chain of causation. If the chain of causation is broken by a *novus actus interveniens*, so that the old cause goes and a new one is substituted for it, that is a new act which gives a fresh origin to the after consequences.

The tests to be applied in determining this question appear to be: "Is the workman's condition, of which he is complaining, in fact due to the original injury, whatever it was, aggravated by infection or disease?" or "Is his condition in fact due to the infection or disease quite independent of the original injury?" An affirmative answer to the former question entitles the applicant to succeed, but an affirmative answer to the latter question shows that the injury is due to *novus actus interveniens* and the applicant cannot succeed.

If a man dies under an anæsthetic administered for the purpose of carrying out an operation rendered necessary by his accident, the death is due to the accident, but if, having sustained an accident, the anæsthetic is administered for a purpose unconnected with that accident and the workman dies under that anæsthetic, then his death is not due to the accident.

The necessity for a careful investigation of the circumstances of an accident will be apparent from this incident.

A workman had broken his leg and was receiving compensation. Whilst convalescing on crutches, he broke it again in the same place. A medical report on the second accident was obtained which ascribed it to the man's slipping on linoleum whilst walking on crutches and it was almost decided to include the recovery from the second break in the man's compensation, when it was noticed that the doctor had not stated where the second accident had occurred. Further inquiries were instituted and it was found that this accident had happened whilst the workman was visiting the local hotel after hours. The claim, as you may well imagine, was compromised.

Incapacity for Work.

Again in most cases the answer to the question of incapacity for work is simple. It is in the stages where the workman should ordinarily speaking be on the high road to a return to work, that most difficulties occur. That bugbear of all lawyers, neurasthenia, frequently interposes to prevent a return to work.

In dealing with these neurasthenic persons, medical men should take into account the factors of: (i) dearth of employment in industry, (ii) the right of a workman to a lump sum after his weekly compensation has been continued for six months, (iii) the fear that if he returns to work he will lose his right to compensation, if his incapacity recurs as a result of the accident.

If the third factor is found to exist, it should be explained to the workman that, if he goes back to work without having signed a formal discharge of all liability, he will be entitled to compensation if his incapacity returns as a result of the accident

and as regards the second factor that it may be advisable for him not to release his rights for a lump sum, if there is a likelihood of his incapacity returning.

With regard to neurasthenic persons, the law applies the test of the average reasonable man and requires an injured workman to exercise normal will power. If as a result of the accident he has not got this will power, then, of course, he is not expected to exercise it. Medical men should be very chary in telling workmen that they will never be fit again or never be able to work as well as they did before or in any other way destroying their hopes of ultimate recovery. One frequently meets with difficulty in settling cases in which the medical report is favourable to a return to work, by being faced with a statement by the workman that Dr. So-and-so said he would never be able to do hard work again.

Extent and Duration of Incapacity and Wage Earning Capacity.

The principles which should guide a medical man in finding answers to the extent and duration of incapacity and wage-earning capacity have been touched upon whilst other matters were dealt with. In arriving at a decision, the medical man should proceed warily and act judiciously. He should endeavour to ascertain the outlook on the future of the workman, his capabilities as regards all forms of employment and his mental and moral equipment. On the final report of the doctors may depend the payment of large sums of money.

With this consideration in view, he should endeavour to form an estimate of the industrial potentialities of the workman in every avenue of work open to him. Although owing to a fixed knee a bricklayer may never be able to climb a ladder again, it does not follow that he is permanently incapacitated for all work. The test is: "Can he earn in some suitable employment the same average weekly wages as he was earning prior to the accident?" and not, "Can he work as other men?" One frequently hears doctors express the opinion that a man will never be able to work as he used to or that his efficiency is reduced 35%. These statements are of little value to the insurance company or lawyer charged with the settlement of a claim.

I realize that it is very difficult for medical men to dogmatize on these matters, but I feel that if they keep the principle of workmen's compensation clearly before them, they will be able to arrive at far more accurate and valuable opinions than they otherwise would.

Medical Reports.

I have been asked to say something provocative and probably I shall now supply that deficiency. In my experience medical reports are as a general rule unsatisfactory. They so seldom give what is wanted. This may be and most probably is the fault of the insurance company or of the lawyer seeking the report not putting specific questions. The use of medical terms without a popular explanation of them is one source of difficulty and the failure to appreciate the requirements of a good report is another.

I give as examples of bad reports the following:

The workman is suffering from spondylitis, the result of an accident.

Or

The man's earning capacity is reduced by one-third.

I now give an example of what I consider a good report:

The left eye shows a scar on the outer side of the cornea (or clear part of the eye). The iris forms a transverse pupil and is adherent to the scar in the cornea. On the inner side of the pupil there are remnants of the lens capsule. The lack of vision is due to (1) the scar in the cornea, (2) the adherent pupil, (3) the absence of the lens, (4) the presence of lens capsule limiting the pupil. The man has no central vision, but has peripheral (side) vision which enables him to distinguish objects approaching from the side as shadows.

How much more instructive to the lawyer is this report compared with the following on the same subject matter.

There is a leucoma on the cornea which has given rise to a synechia anterior. The consequent defect in vision will be permanent.

In my own practice I have evolved a list of questions which I generally put to doctors, when calling for a report. These questions necessarily vary according to the type of case calling for report, but in general they run something like this.

1. What is the present condition of the workman?
2. Is that condition likely to improve or remain stationary?
3. Is the workman now able to return to his previous employment or to some other suitable employment? If so, state such employment or employments.
4. If the workman is not now able to return to work of any kind, will he ever be able to do so and, if so, when?
5. Is the workman now able to do light work or, if not now, when, stating in each case the class of light work?
6. Can you suggest any treatment which would improve the workman's condition?

I suggest that in furnishing reports, wherever possible, a diagram should be enclosed. This is especially necessary with regard to hand injuries where compensation is payable for loss of joints. When amputations have taken place, I generally enclose a drawing of the hand and ask the medical man to indicate on that drawing the parts amputated. This leads to more precision than having to consider what is meant by "the first or second phalanges" or "the metacarpal joint."

I have been glad of the opportunity of addressing you, because I consider it of the utmost importance that an intelligent liaison should exist between medical men and lawyers in connexion with workmen's compensation. It should be our united object to do justice to the claim of the workman on the one side and the rights of the employer or insurance company on the other. This can best be achieved by a thorough and sympathetic understanding of the difficulties which we have to surmount.

THE INSURANCE ASPECT OF MEDICAL CERTIFICATES.[1]

By LESLIE K. McDONALD,
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IN an address on the insurance aspect of medical certificates you will appreciate that to an extent both the medical and legal factors are comprehended. An insurance officer must reconcile the report of the accident with the nature of the injury, as certified by the patient's doctor, in order to discover whether the claimant is entitled to compensation.

A casualty insurance office is interested in claims for personal injuries under three classes of its business and their order of importance in claim frequency is: (i) Workmen's compensation, (ii) personal accident, (iii) third party.

Mr. Skipper has placed before you very clearly the nature of the happening required to bring it within the meaning of the expression "personal injury by accident" and thus to establish a claim under the *Workmen's Compensation Act*. Medical men concerned with the examination of injured men who have made claims under the *Workmen's Compensation Act*, are amazed at the number of cases of severe disability and even death which are attributed to the most trifling accidents occurring in the course of employment. Cases frequently come under notice in which the workman's physical condition predisposes him to an injury and, whilst it is true that an employer accepts a workman with all his physical disabilities, in the sense that it is useless to contend that no injury would have occurred but for the man's state of health, yet it must be shown by the claimant that his employment "materially" contributed to the injury. This aspect is fully discussed in the House of Lords case, Clover, Clayton and Company *v.* Hughes, where a workman suffering from an advanced aneurysm of the aorta was doing his work in the ordinary way by tightening a nut with a spanner. This ordinary strain caused a rupture of the aneurysm which resulted in death. The aneurysm was in such an advanced condition that, according to the medical evidence and to the judgement of the County Court Judge, it might have burst while the man was asleep and a very slight exertion or strain would have been sufficient to bring about a rupture. The County Court Judge found that the strain put upon the workman by the exertion of tightening the nut with the spanner caused the rupture of the aneurysm. He therefore held that the workman died from personal injury caused by accident arising out of and in the course of the employment. The judgement was reaffirmed in the Court of Appeal and in the House of Lords. The following extracts from Lord Loreburn's judgement in this case are most instructive on the point:

> It may be said, and was said, that if the Act admits of a claim in the present case, everyone whose disease kills him while he is at work will be entitled to compensation. I do not think so, and for this reason. It may be that the work has not, as a matter of substance, contributed to the accident, though in fact the accident happened while he was working. In each case the arbitrator ought to consider whether, in substance, as far as he can judge on such a matter, the accident came from the disease alone, so that whatever the man had been doing it would probably have come all the same, or whether the employment contributed to it. In other words, did he die from the disease alone or from the disease and employment taken together, looking at it broadly? Looking at it broadly, I say, and free from over nice conjectures: Was it the disease that did it or did the work he was doing help in any material degree? In the present case I might have come to a different conclusion on the facts had I been arbitrator, but I am bound by the findings, if there was evidence to support them.

It should be noted that in England the judge of the first hearing is the final arbiter of questions of fact and if there is any evidence to support his finding, the case cannot be upset on appeal. This case has been commented upon in several other judgements as being an extreme case. Lord Dundas in Spence *v.* Baird, said:

> The appellant's counsel naturally relied upon the aneurysm case, which probably went as far in this region of the law as any case that has yet been decided . . . and it is important to observe that the decision of the majority —and it was a very narrow majority—was expressly put upon the view that there was evidence upon which the County Court Judge, upon a conflict of evidence, was entitled to hold as he did in favour of the workman.

The more one reads these House of Lords decisions, the more one is struck by the sanity behind the law. I might mention that, unlike the English Act, there may be an appeal on questions of law or fact or both, so that judgements which one might say almost border upon perverseness, might very well be upset here on appeal. It should also be noted that under the South Australian *Workmen's Compensation Act* an employer is afforded a measure of relief from having to compensate those who are under any physical or mental incapacity which is likely to render them more prone to accident, or to whom the result of an accident would be specially serious, or to workmen over the age of sixty years. In such cases the maximum liability may be reduced from £700 to £100, subject to an agreement in writing, prior to the accident, of course. So that the remedy for compensating derelicts, whose preexisting disease might figure largely in injuries contributed to by the employment, is to a certain extent in the hands of the employer himself.

In claims under personal accident policies the requirement to establish a claim is that the "insured shall sustain bodily injury caused directly and solely by violent, accidental, external and visible means." Accidents which are due to any physical infirmity or predisposition in the insured are excluded and in this particular there is a distinction between claims made under a personal accident policy and those which are made against an employer under the *Workmen's Compensation Act*.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on March 28, 1929.

Third party insurance involves claims for personal injury which are based upon negligence such as are due to motor car accidents *et cetera*, involving injuries to passengers or pedestrians *et cetera*.

I have explained shortly the circumstances giving rise to claims and it will be seen that they vary considerably, but in so far as medical certificates are concerned, there is one factor common to them all and that is that the certifying doctor should confine himself to a statement of medical facts. An insurance office requires to know: (i) The nature of the injury (with a reasonable amount of precision), (ii) the probable cause, (iii) the estimated duration of disablement.

It is not overestimating it to say that over 90% of the claims made upon an insurance office are settled on this simple form of certificate without challenge and without reexamination by the company's medical officer. Thus it will be seen that we place a great deal of reliance on your certificates and naturally we expect that a medical man will depose fairly and dispassionately to the result of his examination and will not be an advocate for either side.

When the claimant's doctor is in doubt either as to the cause of the injury or the disabling effect of it, he should state sufficient in his certificate to place the insurance company upon inquiry. I should like here to state my strong belief in consultations between the company's medical adviser and the patient's doctor. Justice is more likely to be done all round when the two sides meet and their only concern is a diligent search after the truth. A reputable company does not wish to shirk its responsibility and the claimant on the other hand should not receive more in compensation than he is fairly entitled to. *À propos* of these remarks I would point out that in an English appeal case the judge said:

> I think it is lamentable that the injured man should have put any obstruction to the full medical evidence of his condition being given to the other side. The medical evidence should be at the disposal of each side.

Wherever possible I encourage conference between medical men and I can testify to the satisfactory results that accrue from this procedure, and you will judge of it for yourselves when I tell you that during my term of management of my company, extending over nearly twenty years, we have not litigated upon one single case based upon a conflict of medical testimony.

There is a reluctance on the part of some doctors to state even the nature of the injury, contenting themselves with "injured hand," "injured back" *et cetera*. In regard to injured backs, there is a mutual distrust, on the workman's part from a dread of the unknown and on the insurance company's part from a suspicion born of experience. I do not know whether this reluctance is due to a fear of being involved in court proceedings, but as a legal fact you may perhaps like to know that a medical certificate may not be produced as evidence without the author's being called, when he will have the opportunity of qualifying or amplifying any statement contained in his certificate.

There is a misapprehension on the part of some medical men as to the necessity for a workmen's compensation claimant to produce a medical certificate to his employer. I have before me a circular issued by members of one of your subassociations informing employers and insurance companies that the onus was upon them to provide and pay for all medical certificates. This misapprehension is due to a misunderstanding of two paragraphs in the First Schedule of the *Workmen's Compensation Act*, which read: Paragraph 4.

> Where a workman has given notice of an accident, he shall, if so required by the employer, submit himself for examination by a duly qualified medical practitioner provided and paid by the employer *et cetera*.

Paragraph 14.

> Any workman receiving weekly payments under this Act shall, if so required by the employer, from time to time submit himself for examination by a duly qualified medical practitioner provided and paid by the employer.

The above paragraphs apply in cases where an employer may require an independent medical examination of the workman, in which event the employer chooses his doctor and pays for the examination and it is compulsory for the workman to undergo this examination or otherwise his compensation is suspended. This, however, does not relieve the workman from discharging the onus which is upon him of proving that his incapacity for work is due to personal injury by accident arising out of and in the course of his employment and it is part of the necessary proof that he should produce a medical certificate. It has been put to me in discussion here this evening that nowhere in the act is it stated that a workman has to produce a certificate to an insurance company and upon that point I would inform you that there is no substance in such a contention. The insurance company acts for and on behalf of and in the name of its insured, who is "the employer," and that it is a well-founded law of insurance that the indemnifier stands in the shoes of the indemnified and therefore any rights which the insured has are vested in his insurer.

Some cases, about 10% of the whole, present difficulties in either or both the medical and legal factors and Dr. Corbin and Mr. Skipper have dealt with a number of such in their very interesting addresses. In these special cases the workmen are referred for reexamination by either the insurance company or a solicitor and usually the question for medical opinion is stated. What is required in most cases is information as to: (i) The injured man's present condition; (ii) when there is a degree of permanency in the injury, what improvement may be expected, and the extent of incapacity that will remain; and (iii) whether the injured man will be disabled from following his usual employment and, if so, what suitable employment could he follow, the words "suitable employment" being taken from the *Workmen's Compensation Act*.

The report should not contain an opinion as to the *quantum* of compensation to which it is thought the workman may be entitled, as this is subject to many legal considerations and is a function of the solicitor or the insurance company. I cordially agree with the statement which has been made in the discussion tonight, that it is to be deplored that there is often such a long lapse between the date of injury and the date of medical reexamination, which more often than not is due to an uninformative certificate issued by a workman's doctor in the first instance. Although the first examination may reveal an injury which will cause a lengthy disablement, a certificate is given to the effect that the man will be unable to follow his employment "for a fortnight." This would be overcome, if an indication were given either as to the serious nature of the injury or that the disablement would be likely to last several months.

RÖNTGENOGRAPHIC STUDY OF THE MOVEMENTS OF ABDUCTION AT THE NORMAL SHOULDER JOINT.

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THE mechanism of the movement of abduction of the normal shoulder joint is described in different standard text books of anatomy in various ways. Some indeed, even such well known books as Cunningham,[1] Piersol,[2] Berry,[3] Buchanan[4] and Ewart,[5] make no reference to this particular problem. Others, however, have followed different conventional teachings which were in existence before any aid may have been given to the question by X ray examinations.

Morris[6] states:

Further elevation of the arm beyond the right angle, in the abducted or extended position, is effected by the rotation of the scapula round its own axis by the action of the *trapezius* and *serratus anterior* muscles upon the sternoclavicular and acromioclavicular joints respectively.

This description has been altered in a later edition as follows:[7]

In abduction and adduction, the scapula is fixed and the humerus rolls up and down upon the glenoid fossa; during abduction the head descends until it projects beyond the lower edge of the glenoid cavity and the greater tuberosity impinges against the arch of the acromion; during adduction the head of the humerus descends in its socket, the arm at length reaches the side, and the capsule is completely relaxed.

Parson and Wright[8] state:

At the shoulder joint it is only possible to abduct till the arm forms a right angle with the body; when the arm is raised higher, it will be felt that the movement is due to rotation of the scapula.

Quain[9] states:

Movement about the antero-posterior axis which runs parallel to the glenoid cavity, is, of course, prevented in one direction by the arm coming in contact with the side, but is free in a lateral direction. The abduction resulting from rotation round this axis is in an upward and forward direction and amounts to 91°; but when the arm is raised from the side in the frontal plane of the body, the movement is limited to 64° and the arm cannot be raised to a right angle without a rotation of the scapula and upward movement of the glenoid cavity.

McKendrick and Whittaker[10] repeat the views of some of the foregoing as follows:

Normal shoulder (abducted about 80°). Note the relation of the anatomical neck of the humerus to the lower margin of the glenoid cavity. If further abduction is carried out, the relations are practically unaltered. Further abduction of the limb is accompanied by movement of the scapula and clavicle.

Gray[11] quotes a very old article by Cathcart[12] based on a study of the action of the various muscular groups about the shoulder, which is evidently much nearer the truth than any of the foregoing. Cathcart concludes:

1. That the scapula rotates throughout the whole movement in question, with the exception of a short space at the beginning and at the end.
2. That the humerus moves on the scapula, not only from the hanging position to the horizontal, but also in passing upwards as it approaches the vertical above.
3. That the clavicle moves not only in the second half of the movement, as is rightly described by some, but in the first as well, though to a less extent.

As the movements of abduction lend themselves particularly well to Röntgenographic study throughout the whole range of movements, from the arm touching the side of the body, to the position with the elbow situated vertically above the joint, an examination of the various movements was made as follows:

Skiagrams were taken of three individuals with apparently normal shoulder joints. These included the spinous processes of the upper thoracic vertebræ and both extremities of the clavicle. Tracings of the essential features were made through transparent celluloid paper using Indian ink.

The following lines were drawn:

(*a*) In the median vertical plane through the tips of the spines of the thoracic vertebræ.

(*b*) In the axis of the clavicle, by joining the mid-points of the sterno-clavicular and acromio-clavicular joints respectively.

(*c*) In the vertical diameter of the glenoid cavity by joining the highest and lowest points. As this forms a fixed angle with any other axis of the scapula, it serves readily as an index of the rotation of this bone.

(*d*) In the axis of the humerus, by joining the centre of the head of the humerus with the centre of the medullary cavity at the most distal part included in the view.

The following angles were measured with the aid of a protractor:

(i) Angle of abduction, that is the angle which the axis of the humerus makes with the vertical line (*a*).

(ii) Angle of scapular rotation, the angle made by the vertical diameter of the glenoid cavity (*c*) with the vertical line (*a*).

(iii) Angle of clavicular depression, the angle made by the acromial extremity of the axis of the clavicle (*b*) with the vertical line (*a*). The angle of clavicular elevation is the complement of the angle of clavicular depression.

(iv) Humero-scapular angle, formed by the axis of the humerus (*d*) with the vertical diameter of the glenoid cavity (*c*). This is also obtained by subtracting (ii) from (i).

(v) Scapular-clavicular, formed by vertical diameter of glenoid cavity (*c*) with axis of clavicle (*b*). This is also the complement of the sum of (ii) and (iii).

The figures in the following tables represent the angles obtained in three different subjects.

Position.	Angles in Degrees.						
	1	2	3	4	5	6	7
Subject A.							
(i) Abduction	8	33	45	104	130	176	
(ii) Scapular rotation	11	25	33	39	42	65	
(iii) Clavicular depression	84	72	65	61	51	63	
(iv) Humero-scapular	−3	8	12	65	88	111	
(v) Scapular-clavicular	85	83	82	80	87	62	
Subject B.							
(i) Abduction	4	22	43	83	88	120	157·5
(ii) Scapular rotation	18	24	28	38	37	42	55
(iii) Clavicular depression	70·5	77	70	62	70·5	60	75
(iv) Humero-scapular	−14	−2	15	45	51	78	102·5
(v) Scapular-clavicular	91·5	79	82	80	72·5	60	60
Subject C.							
(i) Abduction	1·5	46	55	93·5	175		
(ii) Scapular rotation	10	24	22	24·5	54		
(iii) Clavicular depression	68	72	74	59	67		
(iv) Humero-scapular	−8·5	22	33	69	121		
(v) Scapular-clavicular	102	84	84	96·5	69		

From the above figures it is perfectly clear that during abduction the humerus rotates in the glenoid cavity in all positions and that in the more elevated positions the rotation is even more pronounced than in the less elevated. The various descriptions given (Parsons and Wright, McKendrick and Whittaker and formerly by Morris) that the humerus ceases to rotate beyond the horizontal position is manifestly erroneous. The amount of such rotation reaches to at least 114° to 129° in each of the three subjects, although Quain[9] has limited such movement in his description to 91° in an antero-lateral direction and only 64° in the lateral.

The clavicular depression tends to diminish or rather the elevation of the clavicle increases somewhat with the movements of abduction, but this is by no means regular or constant.

The scapula also continues to rotate on its long axis in every porgressive movement of abduction, varying from 37° to 54° in the three subjects examined.

In an examination of the dried skeleton with the humerus, scapula and clavicle in position the acromion forms a definite prominence against which the greater tubercle of the humerus appears to impinge when the arm is placed in the horizontal position, and it is probably this fact which is responsible for many of the current incorrect descriptions. The exact mechanism, more particularly the precise relationship of the acromion process and greater tubercle will be the subject of later studies. Unfortunately, the movements of circumduction do not lend themselves readily to similar Röntgenographic study owing to the difficulty of securing lateral views of the shoulder joint.

Meanwhile, it appears that the description of the movements as given by Cathcart[12] in 1884 is correct, if the exception mentioned in the first clause be omitted.

With regard to the up and down movements of the head of the humerus as described by Morris in his more recent work, a close scrutiny of the joint in various positions shows a constant relationship of the centre of the head of the humerus to the glenoid cavity and no such descension or ascension is seen. In fact the centre of the head of the humerus forms the apex of an isosceles triangle with the highest and lowest points of the glenoid cavity. The shape of this triangle is constant in all positions of the joint.

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Reports of Cases.

X RAY DEMONSTRATION OF THE OCULAR BULB.

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KÖHLER in his book on "Röntgenology," as translated into English by Turnbull, 1928, writes on page 282: "It

is only a vivid imagination that can discover the *bulbus oculi* in a Röntgen picture."

Nevertheless, I have frequently noticed the appearance of the optic globe in skiagrams of the head and no doubt many others have noted the same. The accompanying illustration, therefore, is from a skiagram which shows very clearly on both sides symmetrical circular shadows filling up the greater part of the orbital cavities. On inspection of the upper part there appears to be an outer denser ring which probably corresponds to the sclerotic coat, bounded both externally and internally by much less dense rings, the latter probably corresponding to the loose areolar tissue of the capsule of Tenon and the other to the chorioid and retina, whilst the dense uniform shadow within this must represent the vitreous.

As this is a female subject of the age of fifteen years only, it is not due to any special calcareous change noted in various regions with advancing years. Moreover, I have seen similar changes in considerably younger children.

Possibly Köhler has based his statement upon his experience of skiagrams taken without the aid of the Potter-Bucky diaphragm. The one here illustrated is taken with this accessory.

TWO CASES OF CONGENITAL DEFECTS OF THE LUMBAR SPINE.[1]

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CONGENITAL DEFECT OF THE FOURTH LUMBAR NEURAL ARCH.

(D. R. W. COWAN.)

The patient is an adult male, aged twenty-eight years. He came under observation first in May, 1928, complaining of pain in the lower part of the back. The salient points in his history are as follows: About Christmas, 1927, he had a severe illness with shivering, pyrexia, headache and delirium, which lasted for a few days. A lumbar puncture was done without untoward incident and several cubic centimetres of clear fluid under considerable pressure were withdrawn. The puncture was first attempted under local anæsthesia without success and a general anæsthetic had to be given. No further examination of the fluid was made. Immediate relief from the symptoms was experienced and the patient left hospital at the end of a week, apparently well.

He returned to his duties in a country bank and carried on without difficulty until Easter. But he says that he was conscious of a dull pain in his back all this time and had his back rubbed two or three times before Easter. About this time while stooping forward he experienced a very severe pain in the lower part of the back; the pain apparently started quite suddenly and was localized in the lower part of the back. He received medical attention and was in bed ten days. On attempting to get up the pain was very severe again and he was kept in bed for a further period of five weeks. The history during this period was a little indefinite. His local medical attendant states that he had symptoms of meningeal irritation, namely, headache, backache, delirium, pains in the limbs and at times pyrexia. His wife who is a trained nurse, said that he had nothing but severe pains in his back, for which he took excessive amounts of analgesic and sedative drugs. He did not seem to be progressing, so was sent to town in May.

He stood a twelve hours' trip in a motor ambulance over rough roads very well and when seen was in good condition and had no symptoms to suggest meningitis or other severe constitutional disturbance. Beyond a slight rise of temperature, the only abnormal finding was tenderness on pressure over the fourth lumbar spine. There was no swelling or redness or deformity and no evidence of pressure on the cord. The symptoms seemed to be purely local and he had apparently rather severe pain on any movement of the lumbar spine. An X ray examination of the spine revealed a defect in the neural arch of the fourth lumbar vertebra. There was what appeared to be a symmetrical fracture through both laminæ of the fourth lumbar vertebra with about six millimetres (a quarter of an inch) separation between the fragments. The stereoscopic pictures show the defect well. There was no displacement of the vertebral bodies or narrowing of the spinal canal. The bones around looked quite healthy. Two radiographers reported independently that it was a fracture.

In the previous history there is nothing of particular note. He has never suffered from any disability affecting his back. Both of his parents died from pulmonary tuberculosis and in 1918 he was suspected of a tuberculous affection and was sent to the country for eighteen months. He gained 18·9 kilograms (three stone) in weight and has had no recurrence of symptoms except an occasional attack of what is called asthma. In 1920 he had a mild attack of appendicitis. He has suffered from colds and nasal troubles for which he has received attention from the nose and throat specialist. In September, 1927, he was treated with an autogenous vaccine for colds; he had about a dozen injections, none of which caused any severe reaction, and this does not seem to have been connected with his illness at Christmas. His wife suffered from pulmonary tuberculosis, but was well at the time of her marriage and has had no recurrence of symptoms. One child is quite well.

The Wassermann test produced no reaction and there were no abnormal findings in the blood. A tuberculin test gave a well marked general reaction, but there were no focal signs to implicate the spine. Nothing abnormal was found in the urine.

He was kept at rest in bed for six weeks and gradually weaned off his sedative drugs. The pain in his back eased off and he had no constitutional symptoms. Another skiagram was taken and showed exactly the same appearance as the one taken six weeks previously. There was no sign of callus formation or evidence of further bony destruction. About a month later the patient returned to work and has carried on steadily since. His back has not worried him, except that he feels a little stiff when he stoops. No support or local treatment of any sort was used for his back. He was given thyreoid extract and "Tricalcine" for several weeks without doing any apparent harm.

Several interesting points arise for discussion:

1. Is this a congenital defect which we have stumbled on accidentally, or is it a fracture, either traumatic or pathological?

2. If a traumatic fracture, is it possible to conceive of a fracture in that position caused by muscular contraction and why is there no sign of callus formation?

3. If a pathological fracture, what is the nature of the pathological process? Is it conceivable that the lumbar puncture could have been in any way to blame? Is it possibly allied to Kummell's disease, which in its typical form is a delayed crumpling and collapse of a vertebral body following injury?

The condition was originally looked on as being a fracture, the cause of which was rather a mystery. But in the light of later information it seems probable that the true explanation of the X ray appearance is that it is due to a congenital defect of the neural arch. This had caused a weakness of the spine and the bending forward was responsible for the pain by slightly separating the synchondrosis or straining the ligamentous structures around.

The case is of some importance from the medico-legal aspect, as it was very difficult to satisfy the lay mind that

[1] Read at a meeting of the South Australian Branch of the British Medical Association on February 28, 1929.

the condition was not directly due to the lumbar puncture. And it might have been difficult to have disproved this contention in a court of law.

EARLY SPONDYLOLISTHESIS.
(L. O. BETTS.)

The patient is a farmer, aged thirty. Thirteen months ago he noticed pain at the back of the right hip after a strenuous day's cricket. He had also been lifting bags of wheat that morning. Since then he has had persistent pain of varying severity radiating down the back of the thigh, sometimes into the calf or as far as the ankle. At one period he had to spend two weeks in bed with it. The pain is relieved by rest and it is accentuated by movement, especially jolting. Riding on a horse stirs it up more than any other exercise. He cannot step across the gutter from the curb, but steps gently into it, one foot at a time, and then moves forward. No history of any serious injury to the back was obtained. He had a normal gait. There was moderate lordosis with a deep groove in the midline over the lumbar portion of the spine. When the finger is run down this, it comes against the fifth lumbar spinous process projecting almost 1·25 centimetres (half an inch) further back than the spinous processes above it. The sacrum was not prominent. Mobility of the spine was normal, except for slight limitation of extension. He complained of slight pain over the lower end of the right sacro-iliac joint on full flexion of the thigh with the leg in extension, with a tender spot in this region. There were no signs of sensory or motor disturbance in the lower limbs.

The skiagram (see Figure III on special plate) shows definite subluxation forwards of the fifth lumbar body about six millimetres (one-quarter of an inch) with a gap in its neural arch between the superior and inferior articular processes. The ends of the bone on each side of the gap appear ragged. The sacrum approaches the horizontal, its upper articular surface being at an angle of 70°. The condition was diagnosed as one of early spondylolisthesis with the usual congenital defect of the

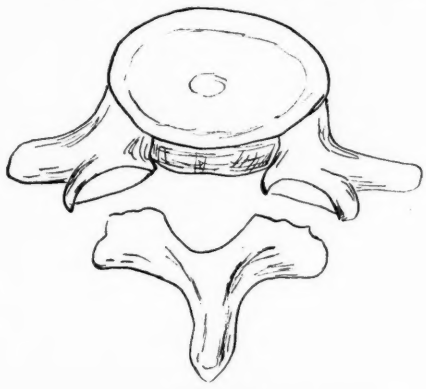

FIGURE I.
Sketch of the type of congenital defect of the lumbar vertebræ existing in the cases reporte dby Drs. Cowan and Betts.

neural arch as the predisposing factor, the actual slipping of the fifth lumbar having started probably with the trauma induced by lifting wheat or playing cricket thirteen months ago. As the patient was an active, robust man who was not prepared to live a quiet, inactive life wearing a support, a bone graft operation was performed to unite the fourth lumbar arch to the fifth and sacrum. At operation the fifth arch was very loose and springy. The arch was not traced out to the lesion, but the periosteal elevator could be felt to slip into the gap. Operation is too recent to report the result.

The case is of interest because early spondylolisthesis is rarely seen. Further, a study of its ætiology provides the answer to the several pertinent questions raised by Dr. Cowan in reference to the case reported by him above. That a defect in the neural arch of the fifth lumbar was the cause of spondylolisthesis was first recognized by Neugebauer[1] in his original writings. There are several types of such defects.

1. Bilateral laminal defects occurring either between the superior and inferior articular processes or at the junction of the pedicles with the body.

2. Incomplete arch of the *spina bifida* type (which causes trouble because of insufficient ligamentous anchorage of the fifth lumbar to the sacrum) with or without.

3. Defective articular facets or facets with definite increased obliquity.

Such abnormalities are quite common. Defects on one or both sides of the arch between the articular processes are present in 4·2% of spines according to Willis.[2] The bilateral defect, as in the cases reported, occurs in 1%. These figures were from an examination made of 740 lumbar spines. The explanation of the development of the laminal defect is still a matter for dispute. Some anatomists state that in these cases the neural arch developes from two centres on each side and that failure of these to fuse produces the defect. Proof of the occurrence of these two centres is lacking. Further, in all such cases of laminal defect examined by Willis fusion of the neuro-central synchondroses and of that in the mid-line forming the spinous process was perfect. As these should fuse at six years and the double centres if present should fuse *in utero*, one would expect that if there were any failure of fusion of the latter, there would be an even more extensive failure of the former.

Although the condition of Dr. Cowan's patient was considered to be a fracture, I think a consideration of the following points should place it in the congenital class.

1. There is no evidence of trauma sufficient to cause a fracture of a normal lamina.

2. The skiagram shows ragged ends to the fragments at the site of the lesion at six weeks, which had not altered at three months after the supposed fracture. In that time there should have been either evidence of bony repair or some alteration of the ends of the fragments. The ragged appearance is typical of congenital laminal defects.

These two cases emphasize the necessity for very careful examination, both clinical and radiological, of patients with chronic pain in the lumbar region or radiating down the thighs. That the defects under discussion predispose to ligamentous strains is supported by the fact that an examination of one hundred unselected films of patients reporting to an orthopædic clinic for pain in the lumbar region, defects were present in 10%. This is a much higher percentage than that found by examination of a series of skeletons. Moreover, radiographs underestimate the number as they will not show many of the laminal defects.

References.

[1] F. Neugebauer: "*Spondylolisthesis et sponyizin*," "New Sydenham Society Selected Monographs," 1885, pages 1-64.
[2] A. Willis: "Lumbo-sacral Vertebral Column in Man: Its Stability of Form and Function," *American Journal of Anatomy*, 1923-1924, Volume XXXII, page 95.

Reviews.

THE RELIEF OF DEAFNESS.

VINCENT NESFIELD has written a book of his own experiences in attempting to alleviate deafness of all types.[1]

In an introduction he admits that his views are unorthodox. Chapters on anatomy and physiology of the ear and the nature of deafness are orthodox and add nothing to our

[1] "Deafness and its Alleviation," by Vincent Nesfield, F.R.C.S.; 1928. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 90, with illustrations. Price: 7s. 6d. net.

present knowledge. "The aim of all remedial methods for middle ear deafness is to reestablish free communication between the atmosphere and the tympanum." The usual method of inflating the middle ear cavity through the Eustachian tube is condemned on the ground that it tends to force the obstructing matter in the Eustachian tube into the middle ear cavity. Instead of Eustachian inflation the author employs a modified mastoid operation in all cases; light general anæsthesia and an 0·5% "Phenolaine" solution injection are used. The bone is of ivory consistency in these cases. A channel parallel to the external auditory canal to the attic is made through the usual mastoid incision. As no hammer is to be used, it is not clear how this dense bone is expeditiously removed by a gouge alone. A warning is issued against injury to the external jugular vein when obviously the lateral sinus is meant. Portion of the bridge is then removed and a flap cut from the posterior aspect of the meatal wall. The mastoid incision is closed and a tube inserted into the attic from the external auditory meatus. Later the tube is removed and tight packing, changed daily for a month, is substituted. The end result is stated to be a subsidiary cavity, lined by mucous membrane and a thin parchment-like membrane closing this cavity at the area of the meatal flap. This membrane has to be incised two to four times a year subsequently to allow aeration of the cavity if deafness recurs. Regarding results the author first states that in 500 patients operated upon no complications occurred and later that the results in over 300 patients treated are hard to determine accurately.

In no instance has the deafness been made worse, in two patients with deafness of short duration (two years) return to normal hearing is claimed. Several cases are reviewed in a general way, but no accurate tests of hearing were apparently carried out before or after treatment. The operation is also claimed to benefit patients suffering from oto-sclerosis and nerve deafness, the author considering that the cochlea has powers of recovery if middle ear health is restored. In an appendix the author describes his treatment for Eustachian catarrh. In this he advocates Eustachian inflation, a treatment he condemned in previous pages. It is appreciated that the author is earnest in trying to alleviate a very distressing disease, but we should have wished for a more accurate record of results. Further, knowing how hard it is to prevent closure of operation cavities in this area, most surgeons would like to see the author's technique carried out before attempting the operation.

A YEAR BOOK ON GENERAL SURGERY.

IN these times there is no lack of a type of book designed to supply the busy practitioner with a condensed *résumé* of the recent advances in his professional work. The work under review, on general surgery, edited by Evarts A. Graham,[1] belongs to an ambitious American programme, known as the "Practical Medicine Series," in which the year's progress in medicine and surgery is reviewed in no less than eight volumes. The size of this book (it contains eight hundred pages) makes more obvious than usual the disadvantages attending the study of such works.

The chief defect undoubtedly lies in the almost complete lack of comment on or criticism of the many conflicting statements quoted therein.

The size of this book allows for the reprinting of many hundreds of articles and case reports, yet the influence, as far as we can judge, of the editor, is noticeable only in that an increased amount of space is devoted to those subjects in which he is known to be specially interested, such as the surgery of the chest and gall bladder.

Otherwise there is scarcely any attempt to balance the many statements, often discordant and, we should judge, of very varying authority, with which the book abounds.

The second criticism lies in the surely unnecessary lack of grammatical accuracy in the text. A little more care expended in composing sentences that could be parsed, would have made the book more readable. There can be no excuse for such a sentence as this, written on the subject of chronic arthritis: "Every case . . . . they put through a thorough investigation for foci of infection, but we don't find them in a good many, and unless the joint is immobilised . . . . he is going to continue to have his painful joint."

But there is a very definite credit side to this book. Practically every important article and case report appearing during the previous year is more or less extensively reported and information can be rapidly obtained which would otherwise necessitate much laborious searching of files. With the reservations mentioned, the book can be recommended as a useful work of reference to the most recent advances in the field of general surgery.

[1] "The Practical Medicine Series, Comprising Eight Volumes on the Year's Progress in Medicine and Surgery," Under the General Editorial Charge of Charles L. Mix, A.M., M.D.; General Surgery: 1928. Chicago: The Year Book Publishers. Crown 8vo., pp. 800, with illustrations.

NERVOUS DISEASES.

THE text book of nervous diseases by Dr. Neustaedter, of New York, differs from others in that the consideration of each disease begins with symptomatology.[1] Notwithstanding that this departure from tradition is emphasized as likely to be useful, we fear many readers may take it as merely a case of putting the cart before the horse. Certainly at the bedside the practitioner or student witnesses signs and symptoms, almost as certainly he also hears a story; if wise, he hears the story first. Usually the story deals with ætiology, therefore its place is first.

The tradition of devoting many pages of a text book to methods of neurological examination is one from which the author might more reasonably have departed, because not only does the existence of several excellent handbooks on methods make these pages redundant, but they occupy space which might have been more profitably used in the body of the book.

In the descriptive chapters the whole field of nervous disease is covered. Chapters deserving special mention are those on infantile paralysis (the Heine-Medin disease), acute encephalitis and diseases marked by tremor, all of which have been specially investigated by the author. Concerning infantile paralysis it may be noted that the author in collaboration with Dr. Banzhaf has prepared an antigen stated to give a reaction with the cerebro-spinal fluid of both infantile paralysis and acute encephalitis. This is put forward as a ground for assuming that both these diseases are caused by mutants of the one organism. The author and Dr. Banzhaf have also prepared an antipoliomyelitic horse serum, claimed to be completely protective to monkeys as well as beneficial to human sufferers. The statement, however: "In the chronic stage, within a few months after paralyses appeared, four intramuscular injections of the horse serum considerably improved them," may provoke scepticism.

On the whole, the book possesses cardinal qualities, accordingly the reader must be lenient in respect of more than a little slovenliness in writing and inaccuracy of language unbecoming as these are. Also, in consideration of the breadth of the subject, he may overlook erroneous remarks such as these, "the lesion for astereognosis is in the supramarginal gyrus," when most authorities place it in the postcentral gyrus; "multiple sclerosis in childhood may be mistaken for Friedreich's disease," the fact being that multiple sclerosis is practically unknown in childhood; and "the intellect in the majority of cases of Little's disease is unimpaired," when the reverse is the case.

The numerous illustrations, most of them original, merit high praise and the publishers have performed their part admirably.

[1] "Textbook of Clinical Neurology for Students and Practitioners," by M. Neustaedter, M.D., Ph.D., with an introduction by Edward D. Fisher, M.D.; 1929. Philadelphia: F. A. Davis Company. Royal 8vo., pp. 622, with illustrations. Price: $6.00 net.

The Medical Journal of Australia

SATURDAY, JULY 27, 1929.

The Prevention of the Spread of Syphilis.

IN the present issue we publish the second part of the Beattie Smith lectures delivered by Dr. Reginald S. Ellery on the pathology and clinical manifestations of neurosyphilis. He has marshalled his facts in a manner calculated to awaken the admiration as well as the approval of the medical profession throughout the Commonwealth. In the course of his lectures he has reminded his readers of the appalling consequences that not infrequently follow a syphilitic infection and he has given evidence in support of the fact that no syphilitic can be guaranteed against the risk of eventual involvement of the central nervous system and of the mental functions of the brain. Folly in youth may result in disaster in middle age; it may give rise to the gravest disabilities in the next generation; it may leave a trail of sadness and misery. The problem of its prevention is one of the most difficult that the hygienist has to face and one of the most important from a national point of view. Like all other infective diseases syphilis should be combated by the whole of the medical profession, not because the individual who contracts it, is particularly deserving of pity and of help, but because he constitutes a danger to those with whom he comes in contact, and it is almost impossible to presage the extent of harm that may result from an infection.

Years ago the subject of venereal diseases was wrapped in silence and mystery as far as the public was concerned. It was regarded as a matter that was so unsavoury that decent men and women should be unaware of its very existence. The teachers of religion avoided it as far as possible and when they felt constrained to make some reference to it, they cloaked their words so effectually that their lessons were largely misunderstood. In recent years all this has been changed. Instead of what is often spoken of as the conspiracy of silence there has grown up a crude outspokenness. Sex talks, sex books, sex films, sex plays have been thrust on a receptive public and have tended to stimulate the imagination, to increase licentiousness and to concentrate attention on this unpleasant but important subject. Sex education is often given to children of tender years in groups. A few learn the significance of sex; the majority are able to continue the lesson and to inform the teacher in intimate detail of those things sexual that he would be unwilling to impart to a child. Education, warnings of the risk of infection, preaching that venereal diseases may lead to irreparable damage to the person infected, to his family and to his future progeny have failed to reduce the incidence of infection. The appeal to youth on the ground of ethics and of religious tenets has also failed. Neither of these methods is likely to succeed in the future, since the public has been told that preventive measures can be applied shortly after promiscuous intercourse with good prospect of success. The youth of today no longer believes in the tales of risk and of disaster. The provision of prophylactic centres where those who have exposed themselves to the risk of infection, may receive preventive treatment free of charge has been of limited use. Many refuse to take advantage of the centres; some feel ashamed of themselves and are not prepared to let anyone know of their folly; some care nothing for the risk, they know of the danger and are improvident; some indulge when under the influence of alcohol and do not realize what they should do. Many seek prophylactic treatment from unqualified persons, from pharmacists and from uneducated quacks. The treatment they receive is often devoid of all prophylactic action. It must therefore be concluded that the provision of public centres for preventive treatment by itself will not suffice to eradicate syphilis.

Modern Australian legislation has made a substantial advance towards the prevention of syphilis. Unfortunately the law is not applied in its entirety in the larger States of Australia. Compulsory anonymous notification to the competent authority is the first essential step. The next step is the notification by name when the person fails to submit himself or herself to treatment until no longer

capable of conveying infection to others. The third step is the relentless prosecution and conviction of persons who have failed to submit to treatment. In this social position, personal influence or other considerations must be ignored. The fourth step is the prosecution and conviction of persons other than registered medical practitioners for treating people for venereal diseases. The fifth step is the severe punishment of anyone who knowingly infects another person. The law holds out promise of a gradual reduction in the number of infections, but this can be effected only if its provisions are enforced under all circumstances. The assistance of the whole medical profession is needed and this assistance to be of value must be whole hearted. Energetic steps should be taken to trace those who fail to report for treatment. The giving of a false name and address is said to be a frequent expedient of those who intend to evade the law. It should be possible for the police to trace many of these defaulters and when they are caught, the sentence should be exemplary. We admit that the world cannot be made moral by legislation; neither is the law powerful enough to eradicate crime. But a great deal can be accomplished if the irresponsible people who leave a trail of disease in their wake, were hunted down and placed under restraint until they realized the enormousness of their offence. Prosecutions of this kind would have an undoubted deterrent effect on the careless and the desperate.

Current Comment.

GENERAL PARALYSIS OF THE INSANE.

IN this week's issue there appears the second of the Beattie Smith lectures, by R. S. Ellery. The first lecture was published last week. The medical profession of the Commonwealth is under a debt to Ellery for his clear presentation of the facts of neurosyphilis and of the doctrines which he upholds. We recommend our readers to go carefully through the lectures and to take stock of the situation. It will be obvious at once that, though definite progress has been made, a great deal of ground has yet to be covered. The causative agent, the *Spirochæta pallida*, is known and can be recognized. Drugs are available which will kill the spirochæte. The latter, however, becomes entrenched in the depths of the cells of the central nervous system and is able on this account to resist the attack by the lethal agent. The part played in recent years by the production of a malarial infection was discussed in these columns on April 13, 1929, in the light of work done by R. B. Wilson and a tentative explanation was offered as a working hypothesis. It may enhance the value of Ellery's contribution, if reference be made to a discussion which took place recently before the Royal Society of Medicine.[1]

The discussion centred round the question of prognosis and treatment of general paralysis of the insane. In a discussion on the value of a certain method of treatment the question of diagnosis must be raised. Ellery points out that old entities such as *tabes dorsalis*, syphilitic hemiplegia, syphilitic epilepsy and paranoia cannot be placed in watertight compartments and in the discussion referred to, John Brander said that a diagnosis of general paralysis was that which was most likely to provoke difference of opinion. He also drew attention to the fact that for some years there has been a tendency to associate under the title, general paralysis, both the classical forms and the so-called neurological or mixed types. This has been done largely on the basis of the serological findings which may be very similar in the two groups. Previously the neurological type was described as insanity with a gross brain lesion. In this connexion it may be stated that it is more logical and more correct to classify diseases from the pathological than from the purely clinical standpoint. The basic lesion in neurosyphilis is vascular and secondary changes occur in the nerve cells. Ellery points out that our conception of neurosyphilis must take cognizance of two factors in the ætiological equation, the individual's basic constitution and the exogenous factor consisting of the location and virulence of the spirochætal infection. Different combinations of these factors will result in different clinical manifestations. Thus general paralysis and *tabes dorsalis*, though clinically diverse, are really neurosyphilitic variations. A wider divergence may be found, for example, between these conditions and syphilitic hemiplegia. General paralysis must always be thought of as a form of neurosyphilis. This is the present day tendency and it is not surprising that in these circumstances there has sometimes been confusion in assessing the value of malarial treatment in general paralysis. It is not to be expected that malarial treatment will be equally efficacious in all forms of neurosyphilis. It might be, if treatment were undertaken in every instance at an equally early stage. In general paralysis the psychic manifestations appear early in the disease before gross paralysis has become manifest. This is not so true of syphilitic hemiplegia or even of *tabes dorsalis*. The result is that, if malarial treatment is undertaken vigorously when the psychic symptoms first appear, there is reasonable hope that the spirochætes may be destroyed before permanent damage, resulting in gross paralysis, has been done. Thus a second con-

[1] *The Proceedings of the Royal Society of Medicine*, April, 1929.

clusion may be drawn—that not only must general paralysis always be regarded as a form of neurosyphilis, but as far as prognosis is concerned, great diagnostic ability is necessary to allow an observer to judge of the likelihood of malarial treatment benefiting the patient. Ellery states that often more refined methods and prolonged experience are necessary. J. Purves-Stewart holds that a diagnosis should not be made on serological grounds alone; the clinical diagnosis must always be established.

Ellery's views on the form of treatment coincide with those of most other observers. Malarial therapy should be used in suitable patients, but reliance should not be placed on it alone. Ellery believes that it should be followed either by mercury and iodine or by intraspinal injections of "Salvarsanized" serum. He also enumerates carefully the danger signals during treatment. Purves-Stewart lays great stress on supplementary treatment by "Salvarsanized" serum. He quotes figures from his private notes. Five patients were treated by "Salvarsanized" serum alone, six were treated by malaria alone and twenty-four were treated by malaria and intracisternal injections of "Salvarsanized" serum. The results in the last named group are regarded by Purves-Stewart as being as encouraging as any others that have been published. Of the twenty-four patients six died, two manifested no improvement, three manifested temporary improvement, four were alive two to six years later, but had relapsed mentally, two were free from symptoms two years later and upwards, two were free from symptoms three years later and upwards and five were free from symptoms four years later and upwards. A modification of malarial treatment was reported at the Royal Society's meeting by F. H. Stewart. Stewart induces short two-day courses of malaria and repeats them at intervals of a couple of months. Purves-Stewart thinks that this might be difficult outside a mental hospital, but not in a mental hospital where time is no object. Another suggestion, made by W. H. B. Stoddart, is the giving of hexamine together with the induction of malaria. He has used this for many years and claims good results in 17% of patients. He deplored the fact that no mention was made of this method in the discussion. He pointed out that hexamine formed formalin in the cerebro-spinal fluid and thought that since it killed some organisms, it might kill spirochætes. "Salvarsan" is known to kill spirochætes, it is efficacious in syphilis of the other parts of the body and its influence is manifested in the altered response of the blood serum to the Wassermann test. There is thus no reason for adopting hexamine, unless for some definite reason "Salvarsan" cannot be used. "Tryparsamide" is mentioned by Ellery. While he would wish to be enthusiastic, he is wisely cautious. T. Tennent reported some interesting results from its administration. Among twenty-seven patients treated by "Tryparsamide" alone, a good remission was obtained in five, a moderate remission in nine, two were stationary at the time of the meeting, seven were in mental hospitals and four were dead. Of twenty-three treated by "Tryparsamide" as well as fever eleven had a good remission, five had a moderate remission, five had a stationary condition, one was in hospital and one was dead. It does not appear to be too optimistic to hope for better results in the future with this drug than have been obtained in the past with other arsenical derivatives.

The last point to be considered is that of cure. Here the old subjects of actual cure and clinical cure crop up. Ellery emphasizes the right point of view in Harrison's aphorism: "We can put out the fire, but we cannot rebuild the house." It were better far to drop the use of the word cure and to refer always to the arrest of a disease. In looking at the clinical improvement it is important to remember that normal remissions may be considerable and of moderately long duration. Importance is naturally attached to the serological findings. On the other hand it sometimes happens that there is an improvement in the serological findings without a corresponding improvement in the clinical manifestations. T. Tennent has three patients in whom this has occurred; in spite of serological improvement the conditions must be regarded as stationary. At the Royal Society's meeting divergent views were expressed. On the one hand G. de M. Rudolf holds that no recovery can be claimed unless the patient has shown a remission up to at least the average age of death for the sex and has died from some other complaint. In addition he demands a normal result of *post mortem* examination. Purves-Stewart countered this by remarking that it was rather a hardship for a patient to wait until he was dead before he was accepted as being cured. Purves-Stewart holds that the standard of arrest should comprise the disappearance of the characteristic serological reactions in the blood and cerebro-spinal fluid as well as the remission or disappearance of clinical symptoms. According to this standard very few would have their disease arrested. The persistence of the reaction on the part of the cerebro-spinal fluid was mentioned in our previous discussion on this subject. J. G. Greenfield thinks that the persistence of changes in the cerebro-spinal fluid is due partly to the scavanging process by which the effects of the acute inflammation occurring in the brain after malaria are cleared up and partly to the scar left in the brain by the disease. He holds that too much attention should not be paid to them. Tennent points out that the most consistent change found in the cerebro-spinal fluid as a result of treatment is a reduction of the cell count. This is found in all cases and occurs early in treatment. The globulin content and the response to the Wassermann test are next in the order of improvement. The change in the response to the colloidal gold reaction is the most difficult to obtain. The ideal to be aimed at is, of course, the normal response of the cerebro-spinal fluid to all tests. In the absence of this the progress of the patient can surely be judged in large measure by improvement in response to successive tests.

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

Visual Disturbances Following Severe Hæmorrhage.

A. FUCHS (*Wiener Medizinische Wochenschrift*, November 3, 1928) describes an instance of profound disturbance of vision following severe *ante* and *post partum* hæmorrhage. The patient could just distinguish fingers at one metre. The optic disc was very pale and apparently covered with a filmy veil. The retinal arteries were contracted, while the veins were of normal calibre. No œdema was noted nor any alteration in the field of vision. Following on a blood transfusion of two hundred and fifty cubic centimetres from the husband immediate improvement was noted and the patient could see fingers at three metres. On the following day the pallor of the discs had disappeared and the arteries were normal, while the vision was $^{6}/_{18}$. A second transfusion was given on the fourth day, after which vision improved to $^{6}/_{12}$. Complete restoration of vision followed in six weeks. The author states that profound visual disturbances following hæmorrhage occur only in those who are not in good health before the loss of blood. The author's patient had a large amount of albumin which persisted after delivery. Such a complication is very serious, especially if it occurs immediately after the loss of blood. He quotes statistics of fourteen patients of whom only three regained full vision.

Voluntary Propulsion of the Eyeballs.

H. FERRER (*Vida Nueva*, October 15, 1928) records the case of a Cuban lad, nineteen years of age, who has possessed the ability during the last thirteen years of pushing either eye or both together out of the orbit and retracting them at will. He is of low mentality and has spent his life in the country. He is one of a family of thirteen children. The family history has no features of interest. The movements are effected with surprising ease and are quite under the control of the will. Ferrer thinks that the movements are carried out by voluntary contraction of the superior and inferior oblique muscles, whilst the four recti are in a state of relaxation. The palpebral fissure is widened and the eyeball slips out. The contraction of the *orbicularis* drives it still further. When the palpebral fissure is again opened and the obliques relaxed, the eyeball slips back into place. So far the trick has been practised from time to time for the purposes of terrifying his small brothers and playmates and no harm has resulted, but now that he has achieved a degree of public notoriety he may be tempted to do it more frequently. The probable results will be irreparable lesions in the optic nerve and retina within the space of a few years. Four photographs illustrate the article.

Retinal Hæmorrhage in Hypertension.

I. AND F. LANGE (*Klinische Wochenschrift*, November 25, 1928) have investigated the ocular signs associated with essential hypertension and arteriosclerosis. Of 60 patients with retinal hæmorrhage 57 had a blood pressure above 160 millimetres and in the majority it was above 200 millimetres of mercury. In fifteen of these no signs of arteriosclerosis could be found. The retinal arteries were narrowed in an irregular fashion. In a vessel varying calibres were noted. Compression of the veins where they crossed the arteries was also noticeable. In the remaining patients with signs of arteriosclerosis the same retinal changes were present with the addition of deposits of lime and cholesterin. In every patient with retinal hæmorrhage associated with essential hypertension an increase of blood pressure was noted. Retinal hæmorrhage is therefore a sign of hypertension and not of arteriosclerosis. Subconjunctival hæmorrhages were also present in a few of the patients suffering from essential hæmorrhage. As in cerebral hæmorrhage, retinal hæmorrhage is due to rupture of the small capillaries.

The Removal of Subretinal Cysticercus.

R. SILVA (*American Journal of Ophthalmology*, November, 1928) describes the method of removing subretinal cysticercus and thus saving the eyeball. He reports two cases. The first was that of a woman of thirty-seven, blind in the right eye for ten months. Examination revealed a detached retina and to the temporal side of the disc a bluish grey sphenoidal bulging in the horizontal meridian of the eye. Its upper part was definitely bounded by a curved line with distinct rainbow hues above, behind and in front. Below the centre of the bulging was a yellow spot, the size of the optic disc, wherein were observed movements typical of the cysticercus. The cyst was carefully localized and measured by the Skeel-Wooton perimeter and under ether anæsthesia the conjunctiva was excised and the external retina divided near its insertion. An incision seven to eight millimetres in length was made in the sclera layer by layer sufficient to cause herniation of the chorioid. The vascular coat was then opened and the vesicle could be seen, but was held by a shell of connective tissue to the chorioid and retina. The operator then carefully broke the envelope of the animal, but to his sorrow the cysticercus remained immovable, though the margins of the wound were held apart. A cataract spoon was used to detach the cyst from its connexions and finally the cysticercus was expelled, measuring fourteen millimetres by twelve millimetres. The second case was remarkable in that the cysticercus abandoned its primary sac and began a tunnel three disc diameters long nearer the equator where the second vesicle was formed. The parasite having been located by the Hirschberg method of noting the scotoma in the field, under cocaine, with a small meridional incision of five millimetres, the sclerotic and chorioid were opened by transfixion, the cysticercus, six millimetres in length, delivered itself. Good vision was maintained.

Shrunken Cataracts in Adolescents.

A. KNAPP (*Archives of Ophthalmology*, November, 1928) describes a method of extracting the shrunken remains of congenital cataracts in young people. The operation is usually performed for cosmetic reasons. The pupil is fully dilated and a keratome incision is made just within the limbus where the periphery of the pupil is clear. The keratome after passing through the cornea, perforates the clear capsule and the opening in the capsule is enlarged by lateral incisions. This causes prolapse of vitreous into the anterior chamber and facilitates future steps. With de Wecken's capsule scissors (Esberg's model) the peripheral capsular adhesion is then divided. The shrunken cataract is seized with blunt capsule forceps (Kalt) and cautiously drawn out of the eye. A bead of vitreous may present at the corneal incision; it is cut off with scissors applied on the flat. Two successful operations are recorded with resulting round black pupils. The main point in this operation consists in cutting the broad capsular adhesion before extracting the cataract.

Conjunctival Bridge in Cataract Operations.

C. T. EBER (*American Journal of Ophthalmology*, February, 1929) in the extraction of cataract employs a conjunctival bridge four to six millimetres wide and ten to twelve millimetres long. An injection of "Novocain" and adrenalin at the spot makes the formation of the bridge very simple. Iridectomy is made from the temporal side. To facilitate delivery of the lens the author inserts the jaws of a pair of special forceps under the bridge; when open they receive the expressed lens. The anterior chamber may be irrigated. In the discussion following the reading of Eber's paper A. E. Ewing said that he used Snellen's loop under the bridge for the same purpose. He removed blood and cortex from the wound and often from the anterior chamber by suction of a stream of lotion passing across the wound, a safer procedure than irrigating the anterior chamber.

Paralysis of the Third Nerve.

M. WIENER (*Archives of Ophthalmology*, November, 1928) has operated upon two patients for the disfigurement following third nerve paralysis. His idea was to transplant the superior oblique muscle to the position of the internal rectus. The superior rectus was exposed and the tendon severed four millimetres from its

attachments, a stay suture being inserted into the stump for purposes of retraction. A hook was then passed behind the equator and the insertion of the superior oblique easily exposed. The tendon was then dissected by first stretching between the hooks and then completing the exposure as far as the pulley. A sharp scalpel was slid into the opening of the pulley and the anterior portion slit through. The tendon was severed from the globe, loosened backwards with strabismus scissors, about 1·25 centimetres (half an inch) was cut off from the end and then it was sutured above the insertion of the internal rectus. The external rectus was exposed and lengthened. In two cases promising results were obtained.

OTOLOGY AND LARYNGOLOGY.

Bloodless Tonsil Enucleation.

F. PETER HERMAN (*The Laryngoscope*, August, 1928) details his method of bloodless tonsil enucleation in which he uses a diathermy current in the tonsil snare. The snare is of the fenestrum type with wire loop; distal to the fenestrum are attached seizing forceps. These forceps are insulated, as are also the snare wire and movable carriage for closing the same. The snare is the active electrode and the grasping forceps the dispersing electrode. The tonsil is engaged in the fenestrum and the seizing forceps are closed to make good contact. The current is then induced by a foot switch and the snare is gradually tightened. The current should not be powerful. This method of applying the current means that the tonsil only is affected. In patients with buried tonsils which cannot be engaged in the fenestrum, the seizing forceps are used as before and a diathermy knife is used to dissect out the tonsils by the usual method. Four hundred and thirty-three patients have been dealt with, 190 under general anæsthesia and 243 with local anæsthesia. No hæmorrhage occurred at operation. In two instances secondary hæmorrhage occurred; both of the patients had undertaken extreme physical exertion two days after operation against advice. The author claims that this method gives cleaner and more rapid healing and that the coagulation action of the snare prevents lymphatic absorption from the fossa during the healing process.

Agranulocytic Angina.

J. I. THOMSON (*The Laryngoscope*, June, 1928) reports two cases of agranulocytic angina. This is a rare and obscure condition usually associated with a definite anæmia. The author hopes that further investigation and the reporting of cases may help to establish its ætiology. The first patient, a female aged thirty-four, developed depressive manic psychosis in her late twenties. At that time she had glycosuria, hyperglycæmia and recurring attacks of secondary anæmia. The mental condition had become disturbed and examination of her blood revealed pronounced secondary anæmia. An ulcer was noted on the surface of the upper lip, covered with a grey exudate. This ulcer enlarged rapidly and ulceration also occurred on the tongue and tonsil. Smears from the ulcerations were found to be free of Klebs Löffler bacillus and Vincent's spirillum, but on culture streptococci and *Staphylococci aurei* were grown. Four days later the smears contained fusiform bacilli and spirochætes. In spite of vigorous general and local treatment the ulceration increased, as did also the anæmia and leucopenia. The patient died. The second patient was a man aged fifty-eight years, quite healthy till August, 1924, when he complained of a slight sore throat and chilly sensations. Pallor of the skin and cyanosis were noted and the tonsils and palate were affected by acute inflammation and were covered with greyish membrane. Diphtheritic antitoxin was given and smears were taken. The smears did not contain the Klebs-Löffler bacillus, but contained a pure culture of *Staphylococcus aureus*. A blood count revealed severe secondary anæmia and leucopenia. The membrane continued to spread and dyspnœa developed; the patient collapsed and died on the third day during a tracheotomy operation.

Influenza and Otitis Media.

G. CLAUS (*Klinische Wochenschrift*, October 2, 1928) refers to the frequency with which influenzal attacks are associated with inflammatory conditions of the middle ear. The onset with high temperature and severe pain resembles acute *otitis media*, but in some cases the temperature is not raised, although pain on the affected side is pronounced. Hæmorrhagic or serous exudates may be noted on both sides of the tympanic membrane and cause a bluish black discoloration. Perforation of the membrane in the postero-superior quadrant is characteristic of many infections. Infection occurs *viâ* the Eustachian tube and a variety of organisms can be cultivated. With the occurrence of perforation pain and fever rapidly subside. Occasionally the discharge dries up spontaneously and the perforation closes, but in many instances there is a spread of infection to the mastoid process with all the classical symptoms. In cases of cerebral involvement the tympanic membrane may appear normal, but there is some tenderness over the mastoid process, while rigors and vomiting develope. The treatment of ear complications due to influenza does not differ materially from that of ordinary *otitis media*. Antipyretics are administered and the membrane is closely observed. The quartz lamp can be of considerable value in treatment at this stage. If this be insufficient or in very acute infections with local symptoms paracentesis must be performed. The radical mastoid operation is reserved for those patients with definite involvement of the mastoid and the diagnosis can be assisted by the use of X rays. The prognosis is good for all mild and subacute infections, but grave if cerebral involvement has occurred. Finally all patients with influenza and dengue fever should be carefully watched for aural complications.

New Operation for Deafness.

J. NORVAL WATT (*The Laryngoscope*, November, 1928) describes his operation for relieving deafness. The main factor in the causation of deafness is the obstructed Eustachian tube and the usual methods of treatment are for the most part of only transient benefit. The operation consists of opening the mastoid antrum through the usual incision. The *aditus* entrance is then enlarged and its patency assured by probing. A small gold tube with a double flange at its external end is bent and fitted to run from the skin surface to the *aditus*. This is inserted and the skin surfaces are sewn up entirely, the soft tissues being drawn in between the two flanges. A cap is supplied and used after the serous oozing ceases in a few days. Success is claimed by this mode of treatment.

A New Self-retaining Palate Retractor.

FRANS HASSLINGER (*The Laryngoscope*, August, 1928) describes a new self-retaining palate retractor. This instrument consists of palatal hook articulating with the shaft of the instrument. This hook is inclined backward for insertion by digital pressure on an articulating rod in the shaft. When in position, the rod is released and the instrument is kept *in situ* by a bar pressing on the upper jaw externally below the nose. This leaves the operator two hands free for manipulation and enables a larger sized mirror to be used for examination of the naso-pharynx. Local anæsthesia by painting with cocaine should be used before the retractor is applied.

Temporal Muscle Grafts in the Mastoid Operation.

HAROLD KISCH (*The Journal of Laryngology and Otology*, December, 1928) advocates using temporal muscle grafts in the mastoid operation. After an ordinary mastoid operation is performed, the temporal muscle is exposed by retraction and separated from the underlying bone. A tongue of muscle is then cut forward, an attachment being left anteriorly. The temporal fascia is divided vertically to allow the tongue to be drawn down into the mastoid cavity. The wound is then closed. In the radical operation no flaps are cut in the auricular canal. The middle ear and attic areas are dressed daily with a paroleine wick after cleansing. Healing is claimed to be complete and rapid and there is no pain, as is usual after flap cutting procedures.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Melbourne Hospital on May 15, 1929. The meeting took the form of a series of clinical demonstrations by members of the honorary staff.

Polyserostitis.

DR. KONRAD HILLER showed a female patient, aged thirty-four years, who gave a history that in 1923 she had suffered from a flooding, although she had previously been in good health. In 1924 she had suffered from influenza. After this she complained of dyspnœa with swelling of the ankles and had suffered from ascites and pleural effusion. Cough and sputum had been present. Between 1924 and 1929 she had suffered from several attacks of acute heart failure. Her chest had been aspirated five times and she had become orthopnœic; hæmoptysis had been present. During the recent months she had been giddy and had complained of nocturnal frequency of micturition.

On her admission to hospital on January 17, 1929, signs of pericardial effusion had been present. A pericardial rub had been audible and a triple rhythm had been heard on auscultation. The systolic blood pressure had been 115 and the diastolic pressure 75 millimetres of mercury. Signs of effusion had been present in both the peritoneal and pleural cavities. The liver margin had been palpable ten centimetres (four inches) below the right costal margin. The spleen had been palpable and the ankles swollen. The condition had improved with treatment. Fluid from the left side of the chest had contained lymphocytes and endothelial cells. "Novarsurol" had been given and after the second injection the urinary output had fallen. The patient had been relieved by potassium citrate and "Theocin." She had been discharged from hospital on March 3, 1929, at her own request with fluid in all her serous cavities. During her stay in hospital several special examinations had been made. The serum had failed to react to the Wassermann test. Examination by the electrocardiograph had revealed a left bundle block. The figures obtained by the urea concentration test had been 1·4% and 1·5% and the blood urea had been 29. X ray examination had revealed a wide aortic shadow with pericardial effusion.

The patient had been readmitted on March 22, 1929, in a comatose, cyanosed and stertorous condition. The breath had not been uriniferous. The systolic blood pressure had been 115 and the diastolic pressure 90 millimetres of mercury. The pulse had been regular and its rate 100 in the minute. The urine had contained albumin, but no casts. The chest had contained an enormous pericardial effusion; it had extended up to the second intercostal space on the left side and up to the level of the third rib on the right side, the dulness blending at the sides with the pleural effusion. Ascites had been present as before. The liver margin had extended one hand's breadth below the right costal margin and the spleen had been palpable. A flaccid paralysis had been present on the left side and the plantar reflex had been extensor in type. The ankles had been swollen.

The pericardium had been aspirated and 480 cubic centimetres (sixteen fluid ounces) of straw coloured fluid removed. Endothelial cells had been present. Venesection had been performed and within six hours a left-sided spastic paresis had developed with increased reflexes. This condition had persisted and had been associated with considerable unilateral œdema of the arm and leg. The mental condition had improved somewhat in twelve hours. The pericardium had been aspirated again and 330 cubic centimetres (eleven fluid ounces) of fluid removed. Since that time some effusion had been present in the pericardium with a small amount of effusion into the pleural and peritoneal cavities. The patient was being kept on morphine and hyoscyamus because of pain in the swollen and paralysed limb. Her general condition was not improving.

Stone in the Common Bile Duct.

Dr. Hiller also showed a Chinese, aged fifteen years, who gave a history of having suffered from dysentery while in China between 1923 and 1927. In October, 1927, he had been in the Melbourne Hospital with jaundice, diarrhœa and pain under the right costal margin. At this time the patient's temperature had been 37·8° C. (100° F.) and the liver had been enlarged to 7·5 centimetres (three inches) below the costal margin. The spleen had been palpable. The leucocyte count had varied between 8,000 and 19,000 per cubic millimetre. Ova of *Ascaris lumbricoides* had been found in the stools, but no amœbæ had been discovered. The Van den Bergh test had yielded a biphasic reaction. The patient had been treated by 0·06 gramme (one grain) of emetine every day for twelve days and after this the jaundice and pain had disappeared and neither the liver nor the spleen was palpable.

The patient's present illness had begun in January, 1929. Up to this time he had been quite well and from then until his admission on March 21, 1929, he had suffered from recurrent spasms of pain in the right epigastrium, with intermittent jaundice and sweating, but no diarrhœa. The patient's temperature had been normal, his tongue coated, his liver enlarged five centimetres (two inches) below the costal margin and the spleen not palpable. No abdominal tenderness had been present, but slight icterus of the conjunctivæ had been noted and a trace of bile found in the urine. During the patient's stay in hospital he had suffered from an attack of severe colicky pain an hour after a meal, in the right epigastrium, accompanied by tenderness in the epigastrium. On the following day the liver had been enlarged ten centimetres (four inches) below the costal margin, jaundice had been pronounced and a generalized rash of raised pink spots had been present on the abdomen. The urine had contained bile and the stools had been greyish in colour. The temperature had been 38·3° C. (101° F.) and the leucocytes had numbered 26,000 per cubic millimetre. Pain and vomiting had persisted for two days and had then become less. The liver had gradually become smaller and the jaundice had gradually disappeared. While this attack was subsiding, injections of emetine had been started again. A second attack of intense colicky pain had occurred on April 15, 1929, and had been preceded by a rigor, the temperature rising to 39·3° C. (102·8° F.). On the following day the jaundice had increased, the liver had become much larger and the leucocyte count had been 22,500. The third and most severe attack had occurred on April 19, 1929. On April 25, 1929, a rigor had occurred without pain, but with an increase in jaundice.

On April 30, 1929, surgical operation had been undertaken. A low grade perihepatitis had been found. The common bile duct had been greatly dilated in the supraduodenal portion and had contained a large mass of biliary mud. The site of this mass had corresponded with a mass which was palpable during the third and most severe attack of colic. The gall bladder had been found contracted and fibrous, but with no stones. Cholecystectomy and choledochotomy had been performed with removal of stones from the duct. The common bile duct and the gall bladder had been drained and convalescence had been uneventful. An examination had been made of the biliary mud. It had been found to consist mainly of bilirubin and calcium with a small amount of cholesterol and it had yielded *Bacillus coli communis* on culture.

Carcinoma of the Stomach.

Dr. Hiller's third patient was a man, aged forty-six years, who had been in the Melbourne Hospital from September 28 to November 2, 1928, and who during this time had been regarded as suffering from a chronic ulcer of the stomach. He had given a previous history of five years' duration with epigastric pain two hours after meals accompanied by vomiting. Some relief had been obtained by the administration of alkalis and regulation of the diet. A test meal had revealed a total acidity of 50 and a barium meal a persistent pylorospasm.

The patient had been readmitted on February 27, 1929, with the history that since his previous discharge he had been subject to pain after meals. During the six weeks

prior to admission the pain had become more severe and had been unrelieved by food, alkalis or vomiting. The vomiting had become progressively more frequent. On examination tenderness had been present in the middle line 2·5 centimetres (one inch) above the umbilicus.

At operation on April 4, 1929, a hard infiltrating mass had been found extending from the pylorus ten centimetres (four inches) along the lesser curvature involving both the anterior and posterior portions of the stomach, the latter being adherent to the pancreas. Small firm glands had been found along both curvatures of the stomach and microscopical examination had shown the mass in the stomach to be carcinomatous and the glands affected merely by chronic inflammatory changes. Relief had been obtained by the performance of partial gastrectomy and the posterior Pólya operation. The patient had been discharged on May 3, 1929.

Pellagra.

DR. F. B. LAWTON showed a male patient, aged twenty-two years, who had been admitted on April 22, 1929, with a diagnosis of pellagra. The patient had suffered from pneumonia as a child and from influenza three or four times. Otherwise he had been healthy and he had been normal at school. He had always been very fond of milk puddings and cream, had never cared for meat, but had been fond of eggs and had eaten lots of bread and butter. The patient had been ill for three years. During this time his condition had improved on several occasions, but he had never become really well. He had complained of indigestion after meals and this had consisted of epigastric discomfort and flatulence. He had also suffered from vertigo and salivation at various times. The salivation had been most severe during the early months of 1929. Throughout the illness he had been constipated except for one month, February, 1929, when he had suffered from diarrhœa. At times he had complained of shortness of breath and a slight cough. Dr. Lawton said that in December, 1927, the patient had consulted Dr. H. Pern, of Leongatha, and that Dr. Pern had been good enough to furnish the details of the early stages of the patient's illness. The patient had become very thin and during July, 1928, an X ray examination had been made. It had been reported that an old ulceration of the duodenum was present, that the ulcer had probably healed and that some distortion of the stomach with delayed emptying had been found. It had been noted in December, 1928, that the mental condition of the patient was bad and that he showed no interest in anybody or anything except his stomach. On April 8, 1929, he had returned with an ulceration of the backs of his hands and the back of his neck. It had resembled a severe sunburn and had manifested a peculiar violet discoloration. The ulcers had healed slowly, but the discoloration had remained. There had also been some desquamation. The patient's general condition had become worse. The ulceration had begun early in February, 1929, and about the same time the patient had complained of a dirty and sore tongue and of ulceration of his mouth.

Since the patient's admission to hospital another X ray examination had been made and this had revealed ptosis of the stomach, the greater curvature being 8·75 centimetres (three and a half inches) below the iliac crest. The stomach had been empty in six hours. It had first been thought that the duodenal cap was deformed, but at a second examination it had been found enlarged and apparently regular. There had been stasis at forty-eight hours in a non-tender appendix with the caecum still filled. A test meal had revealed nothing distinctive and a blood examination had revealed a secondary anæmia.

Polyglandular Syndrome.

Dr. Lawton's second patient was a girl, aged nineteen years, who gave a history that in 1925 she had been in the Melbourne Hospital with renal glycosuria. She complained of lack of energy, of liability to infection, of constipation and dysmenorrhœa. The patient was tall, her hands and feet were large, her features were large and the thyreoid gland was palpable. The pulse rate was 70 in the minute. The basal metabolic rate was −26%. X ray examination of the skull had shown the *sella turcica* to be within normal limits. The occular fundi were normal. The bones of the hand were large and no tufting could be seen on the phalanges on X ray examination. The serum had not reacted to the Wassermann test. The patient had been examined by a gynæcologist who had reported that the uterus was acutely antiflexed. A sugar tolerance test had been carried out. The fasting blood sugar was 0·08%; half an hour after the commencement of the test the figure was 0·11%, after one hour 0·12%, after one and a half hours 0·11% and after two hours 0·09%. The urine, passed while the patient was fasting, had contained neither sugar nor albumin, but a trace of acetone. A twenty-four hours' specimen of urine was sugar free.

Thyreoid Crisis.

Dr. Lawton also showed a woman, aged fifty-five years, who had been admitted on March 6, 1929. She had been treated for ten months for exophthalmic goitre by rest and Lugol's solution. She had improved, her pulse rate falling from 120 to 80 and 90 in the minute and her systolic blood pressure from 200 millimetres of mercury to between 130 and 140 millimetres. Two days before admission she had begun to have severe vomiting and diarrhœa with greenish and very fluid stools containing slime and on a few occasions some bright blood. Culture had failed to reveal the presence of pathogenic organisms. Her temperature had varied from 37·1° to 38·6° C. (99° to 101·6° F.). The pulse had been rapid, the rate being about 140 and at times it had been grossly irregular.

On admission the patient's vomiting and diarrhœa had been less severe, but she had been very exhausted. She had given a typical history of a thyreotoxic condition and exophthalmos had been present. She had lost 12·6 kilograms (two stone) in the previous eight months and she had been unable to do any work for six months and for four months had rested practically all day. Her apex beat had been in the sixth intercostal space 11·25 centimetres (two and a half inches) from the midsternal line and one finger's breadth of cardiac dulness had been present on the right side. A systolic murmur had been audible and the cardiac rhythm regular. The thyreoid gland had been slightly enlarged; one nodule had been definitely palpable in the right lobe and there had been a feeling of irregularity in other parts of the gland. Some improvement had occurred, but on March 12, 1929, auricular fibrillation had become manifest. Two days later the rhythm had again become regular. On March 19, 1929, the basal metabolic rate had been + 30%. Severe cystitis had been present and this had responded slowly to treatment, so that a decision to operate had not been made until April 10, 1929. On this day the basal metabolic rate had been + 19%. On April 14 thyreoidectomy had been performed and thereafter considerable improvement had been noted. The pulse rate had gradually fallen until it was in the neighbourhood of 76 to 80 and the patient had been discharged on April 29, taking 0·3 mil (five minims) of Lugol solution three times a day.

Chronic Nephritis.

DR. W. W. S. JOHNSTON showed a girl, aged fifteen years, who had been admitted to hospital in June, 1928, suffering from acute nephritis complicated by perinephric abscess. The patient had given a history of a reduction in the amount of urine for the previous three months and of swelling in the legs for two months. The only points of interest in the past history were doubtful attacks of diphtheria and scarlet fever.

On admission examination had revealed considerable œdema of the legs and face. No enlargement of the heart had been found, the systolic blood pressure had been 125 and the diastolic pressure 90 millimetres of mercury. Albumin in the urine had been large in amount, 0·6% by the Esbach method. The urine had contained a few red cells and leucocytes and many casts, both granular and hyaline. The amount passed every day had varied between 600 and 900 cubic centimetres (twenty to thirty fluid ounces). The

blood urea had been 61 milligrammes per hundred cubic centimetres and the maximum figure obtained by the urea concentration test had been 1·4%. The perinephric abscess had been successfully dealt with and during the next two and a half months the patient had manifested a gradual improvement, despite an interval during which the systolic blood pressure rose to 162 and the diastolic pressure to 108 millimetres of mercury to fall again later to 118 and 80 millimetres. On discharge from the ward at the end of this period the patient had manifested very slight œdema of the face and ankles. At this stage the urine had still contained a large amount of albumin, an occasional red cell, a few leucocytes and many casts, both hyaline and granular. The amount passed during a day had been in the vicinity of 1·2 litres (forty fluid ounces). The blood urea had been 36 milligrammes per hundred cubic centimetres and the maximum figure obtained with the urea concentration test had been 3·6%.

The patient had then been transferred to the out-patient department and treatment here had comprised large doses of thyreoid extract, up to 0·42 gramme (seven grains) every day; a liberal protein diet had been given, but the intake of salt had been limited. The administration of thyreoid extract had been controlled by the estimation of the basal metabolic rate which had remained practically constant at from −5% to −6%, and by the estimation of the blood cholesterol. On March 14 this had been 0·12% and on May 6, 1929, 0.095%. Dr. Johnston pointed out that there had been a steady improvement in the patient's condition, that she looked healthy and had a good colour instead of being flabby and pale. The urine still contained a large amount of albumin, but microscopical examination revealed only a few leucocytes and neither casts nor red blood cells. The daily excretion was in the neighbourhood of 1·8 litres (sixty fluid ounces).

During the discussion it was pointed out that the patient's condition was probably one of nephritis, rather than of pure lipoid nephrosis in view of the fact that the cardio-vascular system had been involved to some extent, as shown by the temporary rise in blood urea and the simultaneous fall in concentrating power of the kidney for urea and by the temporary rise in blood pressure and the presence of some red cells in the urine. Discussion also took place on the treatment advocated by Epstein in such conditions and on the difficulties resulting from the different interpretation of the term nephrosis adopted by different authorities.

Gout.

Dr. Johnston's second patient was a woman, aged sixty-nine years, whose symptoms had commenced six years previously with a typical attack of gout in the right great toe. Her attacks in this region had ceased, but for the past four years she had had almost constant pain in the fingers of both hands. She had also had symptoms referable to the cardio-vascular system, such as dyspnœa, palpitation and giddiness, and she had been told that her blood pressure was high. The patient's father, two paternal uncles and her grandfather all suffered from gout. The disease had attacked her father in the same form in which it had attacked her. She had five sisters all of whom were well.

Examination revealed multiple tophi in the region of the interphalangeal and metacarpro-phalangeal joints. They were typically chalky in appearance and some of them had ulcerated. Old gouty changes were also present in the metatarsal phalangeal joints of each great toe. The heart was somewhat enlarged. The systolic blood pressure was 182 and the diastolic pressure 88 millimetres of mercury, the radial arteries were thickened. The liver was enlarged. The blood uric acid was 6·5 milligrammes per hundred cubic centimetres and the blood urea 42 milligrammes. Skiagrams of all the joints were shown.

Exophthalmos.

Dr. Johnston also showed a woman, aged forty-six years, who gave a history of having noticed puffiness under the eyes three years previously. This condition had gradually become more pronounced until eighteen months before when there had been considerable protrusion of the eyes and œdema of the soft tissues above and below. There had also been some œdema of the legs. During that period the patient had become very short of breath. The past history contained nothing of interest. The patient had been admitted to hospital on October 5, 1927.

On examination at the time of admission there had been no swelling of the thyreoid gland and no X ray evidence of substernal goître. There had been slight tremor and a moderate degree of tachycardia with a pulse rate up to 115 in the minute and some slight sweating of the palms. The basal metabolic rate had been +15%. The heart had been somewhat enlarged. The systolic blood pressure had been 174 and the diastolic pressure 84 millimetres of mercury. The urine had been free of albumin, but had contained a few granular and hyaline casts. Renal function tests had yielded normal results. Partial thyreoidectomy had been suggested, but the patient had been unwilling to undergo operation and a course of X ray treatment had been instituted and four exposures given. On account of the extreme degree of exophthalmos the outer angles of the palpable fissures had been sutured. Dr. Johnston pointed out that the condition of the patient had remained practically stationary for the past eighteen months. She was being shown in the hope that some suggestions might be made as to the ætiology and treatment.

In the course of discussion the opinion was generally expressed that the patient would probably benefit by operative treatment.

Osteitis Deformans.

Dr. Johnston showed X ray films of typical *osteitis deformans* in a patient seventy-eight years of age. The characteristic rarefying central osteitis and the new bone formation beneath the periosteum were well seen together with the woolly appearance of the skull, while the calcification of arteries was also apparent.

Splenic Anæmia and Gall Stones.

DR. S. O. COWEN showed a male patient, aged thirty-four years, who had been admitted with a complaint of jaundice and an abdominal mass which had been present for many years. His legs had been ulcerated for ten years. Although the patient was thirty-four years of age he was greatly undeveloped physically and to a lesser degree mentally. While in hospital he had suffered from an attack of gall stone colic. He had two brothers and two sisters, all apparently quite well. In none of these was the spleen palpable, but one brother had increased fragility of the erythrocytes. The spleen had been enlarged down to the level of the umbilicus and had extended beyond the middle line. Examination of the blood had shown that hæmolysis started at a concentration of 0·65% sodium chloride solution. The bleeding time had been two and a half minutes and the coagulation time thirteen minutes. The erythrocytes had numbered 2,428,000 and the leucocytes 9,000 per cubic millimetre. No autoagglutinins nor autohæmolysins had been found. A delayed positive result had been obtained to the Van den Bergh test. The Wassermann test had failed to yield a reaction. The patient's blood belonged to Group 4.

At operation the spleen had been removed and but few adhesions had been found. A blood transfusion had been given while the patient was on the table. The gall bladder had been found packed with stones, but the patient's condition had not allowed of their removal. After operation the jaundice had disappeared in the course of a few days and he had gradually got better. The ulcers had been treated by grafting. A fortnight after operation the erythrocytes had numbered 4,000,000 per cubic millimetre.

Artificial Pneumothorax in Pulmonary Tuberculosis.

DR. LESLIE HURLEY showed a female patient, aged thirty-eight years, who had been in good health until November, 1928, when she had noticed a severe pain in the epigastrium and lower part of the chest. The pain had lasted about a week and had been made worse by coughing and breathing. At about the same time she had developed a cough which had persisted ever since. At times the cough

had been paroxysmal and followed by vomiting. For the first two months there had been no sputum, but after that sputum had appeared in gradually increasing amounts, until it reached about a cup full daily. There had been considerable loss of appetite and in the past three months she had lost about 9·5 kilograms (one and a half stone) in weight. On examination on April 26, 1929, there had been slight flattening above and below the clavicle and the left side of the chest had not moved so well as the right. Scattered patches of râles, rhonchi and broncho-vesicular breathing had been heard over the whole of the left lung, particularly in the base and in the axilla and in the same regions there had been slight impairment of the percussion note. Over the right lung a few râles and rhonchi had been heard close to the *manubrium sterni* and along the vertebral border of the scapula and the breath sounds in the same regions had shown some loss of vesicular quality. The systolic blood pressure had been 150 and the diastolic 90 millimetres of mercury. The urine had had a specific gravity of 1015 and had contained no albumin or sugar. Tubercle bacilli had been found in the sputum. X ray examination had shown extensive infiltration in the left hilar region and general mottling in the left lung extending to the base. On the right side there had been some increase in density of lung markings radiating out from the hilus, but no definite evidence of active infection had been detected. There had been some fluid at the left base and the left costo-phrenic angle had been obscured.

In view of the extensive disease in the left lung and the presence of at most only slight disease in the opposite lung, it had been decided to induce artificial pneumothorax. On May 3, 1929, 400 cubic centimetres of air had been injected, on May 6 500 cubic centimetres, on May 8 700 cubic centimetres and on May 11 750 cubic centimetres. A skiagram taken on May 14, 1929, revealed almost complete collapse of the left lung.

Dr. Hurley said that the points of interest in the case were: (i) The presence of extensive disease in one lung with little or no disease in the opposite lung, (ii) the presence of definite disease in the basal parts of the left lung with comparatively little change at the apex.

Dr. Leslie Hurley also showed a female, aged twenty-five years, who had been in good health until September, 1927, when during the latter half of her last pregnancy she had begun to feel unduly tired and languid and had complained of general malaise. She had become rapidly and progressively worse and in the following January, during the puerperium, a cough had become noticeable. She had commenced to lose weight and two months later a right-sided pleurisy had occurred. Her sputum had been found to contain tubercle bacilli. She had then gone to a sanatorium, where she remained for three and a half months, but had returned to her home in July last owing to the death of her baby from tuberculous meningitis. Whilst she was under sanatorium treatment, her cough had diminished, she had gained nearly 6·3 kilograms (a stone) in weight and her general health had improved. However, despite complete rest in bed at home her cough and other symptoms had returned almost immediately. On September 17, 1927, she had had a moderately severe hæmoptysis. On examination she had then presented definite signs of a lesion at the base of the right lung—dulness over practically the whole of the lower lobe, with crepitations and râles and amphoric breathing, suggestive of cavitation, with some prolongation of expiration at the right apex and to a lesser extent at the left apex. Her evening temperature had ranged between 37·2° and 37·8° C. (99° and 100° F.), and occasionally it had risen as high as 38·9° C. (102° F.). She had had repeated small hæmoptyses and despite four months' rest in bed had been losing weight and strength. X ray stereoscopic films revealed consolidation at the extreme right base with an irregular area of translucency above this and an apparent fluid level present, suggesting an abscess cavity. The apical lobes appeared free from any tuberculous parenchymatous lesion. Hydatid tests were all without reaction. There being little or no evidence of changes in the left lung, the induction of artificial pneumothorax had been decided upon. On November 13, 1928, 300 cubic centimetres of air had been injected; on November 17, 1928, 500 cubic centimetres, and on November 21, 1928, 800 cubic centimetres and thereafter at gradually lengthening periods 600 to 1,000 cubic centimetres. Radiologically she had been shown to have definite collapse of the right lower lobe, but complete collapse was prevented apparently by a long band of adhesion. Within a few days of the first induction the patient felt better and she has steadily improved since. During the past five months her temperature has never been above 37·2° C. (99° F.), her cough had considerably diminished and she had seen blood in her sputum on one occasion only. During this period she had been walking about.

Dr. Hurley drew attention to the following points of interest: (i) Incidence of symptoms during pregnancy, (ii) the site of the main lesion at the right base, with little or no evidence of apical involvement, (iii) the satisfactory response to the induction of artificial pneumothorax.

Lead Poisoning.

Dr. Hurley also showed a male patient, aged twenty-nine years, by occupation a worker in a lead shot factory. For the past five or six weeks he had felt tired and languid and his appetite had been poor. In the last three weeks he had had a good deal of pain in the abdomen, chiefly in the epigastric region and at times definitely colicky in nature. On examination the skin and mucous membranes were pale and there was a definite blue line at the margin of the gums. The blood examination revealed the following information:

Red cells, per cubic millimetre	3,700,000
Hæmoglobin value	69%
Colour index	0·9
White cells, per cubic millimetre	24,000
Neutrophile polymorphonuclear cells	73%
Eosinophile polymorphonuclear cells	1%
Basophile polymorphonuclear cells	0%
Neutrophile metamyelocytes	12%
Eosinophile metamyelocytes	1%
Myelocytes	3%
Lymphocytes	9%
Monocytes	1%

A slight degree of anicytosis was present and slight polychromasis and a few stippled cells.

The patient was exhibited because he illustrated the fact that the early symptoms in lead poisoning were usually weakness, pallor and colicky abdominal pain.

The patient had at first been placed on a high calcium diet in the form of milk, with, in addition, calcium lactate, one gramme (fifteen grains) three times a day, to promote the fixation of absorbed lead in the tissues. This had been followed by sodium bicarbonate, eight grammes (two drachms) three times a day, to promote excretion. Excretion could also be aided by giving a low calcium diet along with ammonium chloride, eight to ten grammes daily, or dilute phosphoric acid, twenty to twenty-five cubic centimetres every day.

Sodium thiosulphate also aided excretion by detaching the metal from its protein combination and converting it into a sulphide which could be excreted. The usual dose was from 0·3 to 1·0 gramme every day for several days.

Mitral Stenosis.

Dr. Hurley's third patient was a female, aged thirty-nine years, who had been admitted to the out-patient department on May 26, 1928. At the age of twelve she had had a severe attack of chorea, but the past history had been otherwise normal. For the past three years she had had some shortness of breath which had become more pronounced in the last six months. About six months before admission she had had an attack at night when she had had to sit up to get her breath. There had been no other symptoms.

On examination at the time of admission there had been slight œdema of the feet and ankles. The cardiac apex had been in the sixth intercostal space in the mid-axillary

line and there had been three fingers' breadth of right heart dulness. On palpation there had been a presystolic thrill followed by a sharp apical impulse. On auscultation there had been a loud presystolic murmur, a sharp cracking first sound and a doubled second sound. The pulmonic second sound had been loud and accentuated. Under treatment in the out-patient department with digitalis in doses varying from 0·6 to 1·0 mil (ten to fifteen minims) of the tincture, the œdema had disappeared and there had been definite improvement in her symptoms.

Dr. Hurley said that the points of interest in the case were: (i) The typical signs of mitral stenosis, (ii) the definite improvement under digitalis treatment in the absence of fibrillation and without bed rest, (ii) the great displacement of the apex beat.

Blood Films and Charts.

Dr. Hurley also showed a number of blood films and charts illustrating:

1. The blood picture in a case of acute leuchæmia which had been fatal after an illness of only three weeks. The only features for the first fortnight of the illness had been sore throat with slight evening rise in temperature and weakness.

2. Charts showing the size of red blood corpuscles in normal blood and in pernicious and secondary anæmia. These charts had been prepared by the Edinger projection apparatus, the same magnification being used in each case. They illustrated the fact that in pernicious anæmia there was typically an oval macrocytosis, while in secondary anæmia the size of the cells was less than in normal blood. Charts were also shown illustrating the decrease in size of the red cells in pernicious anæmia under liver treatment.

3. Blood films of the other blood diseases were also exhibited.

Sycocis.

DR. R. R. WETTENHALL showed a male patient, aged thirty years, who was suffering from staphylococcal sycocis. The patient was shown in order to illustrate the effects of treatment by ultra-violet light and to show the rapid reduction of the pustular condition. Dr. Wettenhall explained that the combination of epilation by X rays and subsequent ultra-violet irradiation had proved very successful in obstinate cases of this condition.

Keratosis Pilaris.

Dr. Wettenhall's second patient was a widow, aged fifty-four years, who suffered from *keratosis pilaris* of a pronounced degree occurring after the menopause. The appearance of the condition had been preceded by constipation of two years' duration. Extreme irritability was present, especially in the head. The hands and fingers were unaffected. The trunk and limbs were quite rasp-like to palpation and considerable erythema was present over the orbits and the malar prominences. The appearances suggested a toxæmia of gastro-intestinal origin.

Lupus Erythematosus.

Dr. Wettenhall also showed a man, aged forty-two years, who was suffering from *lupus erythematosus* of a very severe degree. The patient was a syphilitic. The lupus had cleared up temporarily, only to recur in spite of the fact that the patient was under antisyphilitic treatment.

Acne Indurata.

Dr. Wettenhall's next patient was a single woman, aged twenty-four years, an epileptic, who was under treatment with "Luminal" and who suffered from severe *acne indurata*. Bromides had not been given for some months.

Rodent Ulcers of the Face.

Dr. Wettenhall's last patient was a male, aged sixty-nine years, who was suffering from multiple rodent ulcers of the face. The condition was responding well to treatment by X rays and radium.

Cardiac Rheumatic Infection.

DR. DOUGLAS THOMAS showed a series of patients who illustrated the late stages of cardiac rheumatic infection. Many of them had been in hospital several times. The patients were accompanied by their electrocardiograms and skiagrams were shown in order to illustrate the degree of cardiac enlargement present. The significance of the various changes in cardiac outline was discussed and it was explained that the films had been taken by the long distance technique (a target film distance of about two metres) in order to avoid distortion.

A male patient, aged nineteen years, had had four acute rheumatic attacks, ten years, nine years, two years and six months previously. The attack two years previously had been accompanied by pneumonia and the attack six months previously by a severe cardiac breakdown, the patient being in hospital for four months. During this attack the patient's temperature had been irregular and had risen as high as 39·4° C. (103° F.) for three months. There had been no joint involvement at any stage. Since his discharge from hospital the patient had been only moderately comfortable. He could walk only a limited distance without becoming short of breath. Examination of the heart revealed that it was considerably enlarged. The apex beat was in the sixth intercostal space fifteen centimetres (six inches) from the middle line. There were two fingers' breadth of cardiac dulness in the third intercostal space on the right side. A diffuse systolic thrill was palpable. At the apex there was a clear, slapping first sound followed by a diastolic sound. Superimposed systolic and diastolic murmurs were audible. At the aortic area long systolic and diastolic murmurs were present. The heart sounds were heard all over the chest and in the axilla a loud systolic murmur was audible. The systolic blood pressure was 142 and the diastolic pressure 40 millimetres of mercury. The liver and spleen were not palpable. Dr. Thomas explained that the patient was regarded as having had a severe pancarditis with both aortic and mitral lesions. His cardiac rhythm was normal.

A male patient, aged fifteen years, had suffered from rheumatic fever two years previously. Soon after this his ankles had become swollen and he had become short of breath. This had improved so that he was able to play football. Six months before the time of the meeting he had suffered from a severe pancarditis and had been in hospital for three months. Since the discharge from hospital he had steadily improved and he was able to take a moderate degree of exercise with comfort. The systolic blood pressure was 120 and the diastolic pressure 55 millimetres of mercury. The skiagram revealed a considerable degree of cardiac enlargement. At the apex the first sound was accentuated. There was no presystolic murmur, but a systolic bruit was present. The second sound was followed by an early diastolic murmur. At the aortic area both systolic and diastolic murmurs could be heard. This boy was regarded as having a double lesion. His cardiac rhythm was regular.

A female patient, aged twenty-four years, was shown. She had suffered from two or three rheumatic attacks. She was not sure when the first of these had occurred, but knew that it had been in early childhood. The last attack had taken place twelve months previously. At this time she had developed wasting of the right leg and though the details were not reliable, the condition suggested that a peripheral embolus had occurred. The patient was an alert, intelligent girl with a very limited exercise response. She could walk with comfort, provided she did not hurry. The skiagram revealed a gross cardiac enlargement and the electrocardiogram confirmed the presence of auricular fibrillation which was controlled by the administration of tincture of digitalis in doses of 1·2 cubic centimetres (twenty minims) every day. The apex beat was in the fifth intercostal space 12·5 centimetres (two and a half inches) from the middle line. There was two fingers' breadth of cardiac dulness on the right side. The first sound was loud at the apex and was followed by a diastolic murmur. The cardiac rhythm was totally irregular and the rate was about 80 in the minute.

A female patient, aged twenty-eight years, had suffered from acute rheumatism eleven years previously and had been in bed for four weeks. She had been married nine years previously and up to that time had been a member of a physical culture class. During pregnancy she had become very short of breath and at seven months the pregnancy had been terminated. The patient had manifested considerable restriction of her exercise ever since. Six months prior to the meeting she had been admitted to hospital and had stayed indoors for two months. She had then been very ill and fibrillation had been present during the whole of her stay in hospital. On one occasion she had been given 0·1 gramme of quinidine followed by 0·4 gramme twice a day. After the first three doses she had vomited the quinidine and the attempt to restore normal rhythm had been discontinued. Since her discharge from hospital she had been taking digitalis continuously and had manifested considerable improvement. She was walking in comfort, but was incapable of doing her own housework. She had a large heart with both a systolic and a diastolic bruit at the aortic area. At the apex there was a loud systolic murmur as well as a short soft diastolic murmur. Fibrillation was still present.

Complete Heart Block.

DR. GEOFFREY A. PENNINGTON showed a male patient, aged sixty-seven years, who had first reported at the hospital in December, 1926, with a history that despite slight dyspnœa on exertion, he had felt well until, after a heavy day's work as a baker six days previously he had suddenly lost consciousness and had fallen. There had been no premonitory symptoms and no pain. He had remained unconscious for about sixty minutes and had recovered while being carried home. Since then there had been dyspnœa on exertion, sufficiently pronounced to prevent him following his occupation and frequently associated with manifestations of congestive failure. There was a past history of frequent attacks of rheumatic pains from the age of twenty until about ten years previously and of gonorrhœa as a boy. In 1926 the pulse rate had been slightly variable and usually about 32 per minute, but the ventricular rate as estimated from an electrocardiogram taken at that time, had been 38 per minute, the auricular being 83 per minute. The ventricular complex also had been abnormal, showing an inverted *T* in Leads 1 and 2. Unfortunately he had not returned after this examination, until February of 1929, reporting then because of increased dyspnœa.

Examination had then disclosed typical complete heart block. The pulse had been regular, full, of good tension, the rate had been 33 to 34 per minute and there had been some palpable thickening of the vessel. The blood pressure in millimetres of mercury had been 180 to 196 systolic, 74 diastolic. There had been visible pulsation in the neck, partly venous, showing a regular auricular rate of 83 per minute and quite irregular in relation to the carotid pulse which was readily visible in what was apparently an aneurysmal dilatation of the right common carotid artery.

The heart had been enlarged, the apex beat being displaced to a position just outside the nipple line. There had been two fingers' breadth of right cardiac dulness in the first and second right intercostal spaces and slightly less in the third and fourth intercostal spaces. The sounds at the apex had not been normal, the first sound being of poor intensity and followed by a blowing systolic bruit. Occasionally a soft muscular note, probably auricular in origin, had been audible. The sounds at the other regions had apparently been normal. There had been persistent crepitations at the bases of the lungs and the liver had been slightly enlarged, but there had been no œdema of the ankles. The tendon reflexes had been present with the exception of the ankle jerks, but joint sense in the great toes had been impaired. The right pupil had been larger than the left, but there had been evidence of old iritis in the left eye. Both pupils had reacted to light and accommodation and the fundi had been normal except for evidence of sclerosis of the retinal arteries. Signs of rheumatoid arthritis had been present in the joints of the hands and feet.

Radiological examination of the chest had confirmed the pressure of cardiac enlargement and revealed slight general dilatation of the aortic arch. The recent electrocardiograph tracing showed that complete heart block still existed, the auricular rate being still 83 per minute and the ventricular 34 per minute. The urinary examination revealed no abnormality and the Wassermann test had yielded no reaction.

Paroxysmal Auricular Fibrillation.

Dr. Pennington also showed a patient, aged fifty-eight years, who had attended first in 1918 complaining of pain over the left anterior portion of the chest, not radiating and independent of exertion. Examination had then disclosed the systolic blood pressure to be 178 millimetres of mercury and the diastolic 98 millimetres. The arteries had been thickened, but the pulse regular. The heart had been enlarged, the apex beat being in the sixth intercostal space 3·1 centimetres (one and a quarter inches) outside the nipple line. There had been slightly increased aortic dulness. A loud rough blowing bruit had been present at the apex beat, occurring late in systole, being separated from the first sound by a definite interval and conducted outwards into the axilla. The aortic second sound had been accentuated. Clinically there had been no evidence of aneurysm, but radiological examination had revealed generalized dilatation of the arch of the aorta. The Wassermann test had yielded no reaction.

Subsequently, dyspnœa on exertion had gradually developed, but the pain had still occurred intermittently and independently of exertion. In 1926, he had commenced to have attacks of orthopnœa, the heart had appeared to have slightly increased in size, frequent extrasystoles had been present and the bruit had still definitely followed the first sound by an interval. The systolic blood pressure had been 185 millimetres of mercury and the diastolic 100 millimetres. The electrocardiogram had been normal.

In 1928 orthopnœa had frequently necessitated hypodermic injections and mild congestive cardiac failure had occurred which improved with rest and digitalis. Since then, the apical bruit had been definitely connected with the first sound, the apex beat being situated in the sixth intercostal space, 15 centimetres (six inches) from the mid-line, the systolic blood pressure varying from 150 to 170 millimetres of mercury and the diastolic pressure had been 90 millimetres.

On November 26, 1928, the patient had complained of a sudden attack of irregular action of the heart, occurring during the preceding night, with increased dyspnœa and auricular fibrillation had been found to be present. He had been given two cubic centimetres (thirty minims) of tincture of digitalis thrice daily and within forty-eight hours the heart rhythm had returned to normal. A second attack of fibrillation had followed cessation of digitalis therapy in April, 1929, again responding rapidly to treatment.

Since then he had been continuing with digitalis and had had good exercise tolerance, though he was still subject to occasional attacks of dyspnœa and to nocturnal attacks of rapid heart action, if he ceased taking digitalis.

Paroxysmal Tachycardia.

Dr. Pennington's third patient was a male, aged seventy-six years, who in 1923 had complained of sudden attacks of palpitation, especially occurring after meals, but liable to occur at any time, for sixty-two years. Each attack had commenced suddenly with a jump and had ceased with a jump, but he had usually been able to tell from a slight irregularity when the paroxysm was about to stop. The attacks had varied in frequency and duration, lasting from a few minutes to twelve hours and the tendency had been towards gradually increasing frequency.

Originally it had been possible to control the attacks by posture, namely, bending forward and compressing the chest laterally. Of recent years nothing had been of any avail. Quinidine had been tried without effect. Dyspnœa on exertion had gradually developed and if the paroxysm was of any considerable duration it had caused restlessness, dyspnœa and considerable distress.

On examination, gross sclerosis of the vessels was present with tortuosity; the systolic blood pressure was 155 and the diastolic 100 millimetres of mercury. The chest manifested gross rhachitic deformity of the pigeon breast type, and the heart was enlarged, the apex beat being in the fifth intercostal space, 13·75 centimetres (five and a half inches) from the mid-line. The area of right cardiac dulness was increased two fingers' breadth and a soft systolic bruit was audible at the apex.

Of interest was the fact that the rapidity of the paroxysm varied, being 200 per minute in 1926 and 218 per minute in 1929. The ventricular complex as seen in electrocardiograph tracings had also changed during this interval.

Ophthalmic Conditions.

DR. LEONARD MITCHELL demonstrated several interesting conditions illustrating the more usual changes encountered in the *fundus oculi*.

His first patient was a man of thirty-seven with chronic nephritis and a systolic blood pressure of 230 and a diastolic pressure of 160 millimetres of mercury. The blood urea was 287 milligrammes per 100 cubic centimetres of blood and the urea concentration 1·10%; the urine was solid with albumin. The retina showed gross arterial thickening and tortuosity with many scattered spots of exudate of the hard type and a commencing star figure in one eye.

Other conditions exemplified included arteriosclerotic retinitis with thrombosis of a branch of the retinal artery, gross papillœdema with hæmorrhages and exudates in a case of cerebral tumour, exudative retinitis of doubtful origin, optic atrophy following papillœdema, primary optic atrophy, pseudo-cupping of the optic disc and syphilitic chorioido-retinitis showing old and recent changes.

Mandibular Cyst.

MR. VICTOR HURLEY showed a patient, aged thirty-one years, who had first been seen on June 16, 1924, and had given a history of "crackling" of the lower jaw on the left side for five months. There had been no pain, but a noticeable swelling was present. There had been no other swellings in any part of the body. The patient had been wearing complete upper and lower artificial dentures, the upper for some years and the lower for a few months. He was a healthy young man, edentulous, with a swelling of the left lower jaw, from 2·5 centimetres (one inch) to the left of the mid-line as far as the coronoid process. There had been crackling over this on pressure applied inside the mouth where the mucous membrane covered a large area deficient in bone. X ray examination had revealed a cyst occupying the area mentioned, with deficient bony wall.

At operation on June 26, 1924, the cyst had been shelled out practically intact. The wound had healed by first intention and the patient had been discharged on July 12, 1924. Microscopically the cyst was seen to have a fibrous wall with curious patches of cells which from their appearance and the way they permeated the tissue spaces were not reconcilable with any inflammatory causation and were therefore to be regarded as more or less sarcomatous in nature (Dr. Mollison).

The patient had returned on January 17, 1929, with a history that he was quite well till six months previously when the cyst recurred with some pain and swelling. X ray pictures taken in the intervening period showed the cavity filling up well. Examination on January 17, 1929, had revealed a similar condition to that in 1924. At operation on January 31, 1929, the cyst had been shelled out completely. The wound had healed well and the patient had been discharged on February 10, 1929.

The specimen contained an oval shaped unilocular cyst measuring 4·2 by 2·2 centimetres. In the fresh state the cyst contents were composed of cholesterol and fatty *débris*. No teeth elements were present. The walls were white, thin and fibrous. A small rupture had occurred during removal. Microscopically the cyst wall was composed of fibrous tissue lined by stratified squamous epithelium. This layer showed some hyperplasia in one area, but for the most part was simple and well formed. The fibrous tissue of the cyst wall was fairly vascular, the vessels being well formed. There was a dense infiltration with round and plasma cells. There was no bone or cementum in the capsule.

The age of the patient suggested a cyst of the adamantinoma group rather than one of the dentigerous group in which the unerupted tooth played such a big part. The history of slow development and the recurrence after removal favoured an adamantinoma.

Mr. Hurley also showed a woman, aged fifty years, who had noticed twelve years previously a small swelling on the left ramus of the mandible. This had gradually increased in size, but had caused no inconvenience. During the last eighteen months there had been a rapid increase in the size of the swelling which had become very prominent both in the inner and outer aspects of the jaw.

No pain, no salivation and no difficulty in speaking had been noticed. All the teeth in the lower jaw had been removed some months previously. Most of them over the swelling had fallen out easily. All upper teeth had been removed some years before.

On the outer side and beneath the left ramus of mandible and extending from angle to about 1·8 centimetres (three-quarters of an inch) behind the *symphysis menti* there had been a firm swelling, uniformly regular and giving egg-shell crackling. On the alveolar aspect there had been a gap in the bone of 1·25 centimetres (half an inch) between the mandible and the cheek; the swelling had been fluctuant. X ray examination had revealed extension of absorption of the mandible with expansion of bone upwards and downwards. The edges had been sharply cut.

At operation on August 8, 1928, the cyst had been removed intact through a small window cut in the mandible on its outer aspect. Microscopical examination of the specimen had revealed a thin epithelial lining with a fibrous tissue wall.

At a subsequent X ray examination on May 9, 1929, the outline of the cyst cavity was no longer obvious and filling in of the bone cavity was noticed.

Cardiospasm.

Mr. Hurley's third patient was a woman, aged fifty-four years, who had complained of indigestion and difficulty in swallowing for years. She had felt food blocked at about the level of the xiphisternum. She had then been in the habit of vomiting, sometimes immediately and sometimes later, but it would always be the last meal taken. As a rule she had then been free for some time from any discomfort. The diet had been confined to liquid foods for the last few months. She had lost much weight and weighed 41·4 kilograms (six stone eight pounds). Previous treatment by œsophagoscopy and attempted passage of bougies (mercury tubes *et cetera*) had failed. It had been impossible to pass anything through into the stomach.

On October 21, 1927, gastrotomy and digital dilatation from below by the method of Mikulicz had been undertaken. On August 18, 1927, a portion had been removed endoscopically for section. No malignant disease had been discovered.

An opaque meal report had been received on November 14, 1927. After six hours half residue had been found in the lower part of the œsophagus and practically half in the stomach. After twenty-four hours a moderate residue had been present in the œsophagus and a little in the stomach. Some of the meal had been found in the terminal part of the ileum and at the splenic flexure. At forty-eight hours it had been found in the caecum and pelvic colon.

Mr. Hurley said that the patient still had about half the residue of a motor meal in the œsophagus at six hours with a residue persisting at twenty-four hours.

On May 7, 1929, the œsophagus had still been considerably dilated with a little delay in emptying, but the meal had steadily gone through into the stomach. The patient's weight was 69·3 kilograms (eleven stone).

Carcinoma of the Rectum.

Mr. Hurley also showed a woman, aged forty-one years, who had noticed twelve months before admission that her motions were streaked with bright blood. The amount of bleeding had increased and she had passed about one cupful a day.

For eight months she had been defæcating four times a day and had never felt satisfied after any of the motions. When she awoke the desire had been particularly urgent and she had passed blood and slime. At other times she had passed fæcal material.

For three months she had noticed some hard masses protruding from the anus. She had had burning pain in her back passage, when the bowels were open. She had complained of loss of energy and loss of appetite. Secondary anæmia had been present. She had lost 3·1 kilograms (seven pounds) in weight in twelve months.

On examination she had been very pale. Rectal examination had revealed small warty, hard masses six millimetres (quarter of an inch) long protruding from the anus. Hard, nodular masses had projected into the lumen from the anus upwards for 5·0 to 7·5 centimetres (two to three inches). Examination had caused much discomfort and had been very painful. The growth had involved the whole circumference of the bowel.

At operation on November 7, 1928, rectal examination had revealed that the growth extended upwards for five centimetres (two inches). The posterior wall to the sacrum had been clear. The posterior vaginal wall had been involved. Colostomy had been performed.

On November 21, 1929, the Lockhart Mummery operation had been performed under ethylene anæsthesia, the lower twenty centimetres (eight inches) of the rectum with the posterior vaginal wall being removed. Recovery had been uneventful.

Mr. Hurley said that Dr. Mollison had reported that microscopical section revealed columnar carcinoma adjacent to the squamous epithelial surface.

Hydatid Disease.

Mr. Hurley also showed an elderly man who had been admitted to hospital on January 24, 1929. He said that the case illustrated the importance of correct diagnosis in an elderly person with a tumour and wasting. The patient had noticed a lump in the epigastrium four weeks prior to admission. This had been his only symptom. Examination had revealed a mass in the costal angle which moved with respiration. The mass had been hard and irregular and the edge of the liver had been felt below it and moving at the same rate. Signs of cavitation had been present in the apex of the left lung. The Casoni test had been applied. A positive response had been obtained in the immediate test and no reaction had followed the delayed test. The control had been normal. X ray examination had revealed the presence of a calcified cyst 11·25 centimetres (four and a half inches) in diameter in the upper part of the abdomen. The cyst had been situated chiefly to the left side and just crossing the middle line; it had been situated more in an anterior than in a posterior direction close to the diaphragm. The complement deviation test for hydatid disease had yielded a reaction.

On January 30, 1929, surgical operation had been performed. A right upper paramedian incision had been made. The tumour had presented. On incision of the tumour greenish degenerated hydatid cysts had been revealed. These had been scooped out and the cavity plugged with formalin. On February 8, 1929, the patient had suffered from broncho-pneumonia. Tubercle bacilli had been found in the sputum. The patient had subsequently been discharged.

Fracture of the Femur.

Mr. Hurley's last patient was shown in order to illustrate the efficiency of the application of traction by a caliper to the condyles of the femur when the usual method with adhesive strapping and so forth had failed to correct the deformity. The patient, a man of thirty-eight years of age, had been thrown out of a motor car and had fractured the left femur just below the junction of its lower and middle thirds. Posterior displacement of the lower fragment had been present and the limb had been shortened 1·25 to 2·5 centimetres (a half to one inch). The foot had not been everted. The limb had been put up on a Thomas's knee splint with extension. Three days later the position had not been good and the extension had been increased; a pad of wool had been used to produce anterior bowing. On November 23, 1928, an ice-tong caliper had been applied to the condyles of the femur with extension weights of 6·75 kilogram (fifteen pounds). The position had been almost perfect. The patient had been fairly comfortable, but had complained a little. On December 4, 1928, some separation of the fragment had been noted and the extension weights had been reduced by 2·25 kilograms (five pounds). On December 24 the ice-tong caliper had been removed and on February 8, 1929, all extension had been removed from the leg. On February 14, 1929, the patient had been supplied with a walking caliper and had been allowed out of bed.

(To be continued.)

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE Executive Committee of the third session of the Australasian Medical Congress (British Medical Association) informs us that the full programmes of the sections and of the Congress itself are nearing completion. Attention is drawn to the full meeting of Congress on Tuesday, September 3, 1929, at 9.30 a.m., at which there will be a debate, the title of which is given as: "What is Being Done in Australia Towards Cancer Research?" This was announced in our issue of July 20, 1929. At 12.30 p.m. of the same day the official photograph of members attending the session will be taken.

Section of Neurology and Psychiatry.

The meetings of the Section of Neurology and Psychiatry will be held on Tuesday, September 3, 1929, in the practical physiology class room in the Medical School.

Tuesday, September 3, 1929.

2 p.m.—Address by the President of the Section, Dr. W. Ernest Jones.

2.30 to 5 p.m.—"The Pyrexial Treatment of Nervous and Mental Disorders, with Special Reference to the Teratment of General Paralysis of the Insane by Malaria": Dr. Clifford Henry, of Callan Park, Dr. R. Ellery, of Mont Park, Dr. J. Bentley, of Claremont, Dr. E. J. T. Thompson, of Claremont, Dr. R. Gwyn Williams, of Claremont, and Dr. J. Bostock, of Brisbane. A bioscope film illustrating patients undergoing treatment by malarial infection will be exhibited.

"The Use of 'Tryparsamide' in the Treatment of General Paralysis of the Insane," by Dr. E. T. Hilliard, of Orange.

7 p.m.—Dinner by the members of the Section of Neurology and Psychiatry of the New South Wales Branch of the British Medical Association to the visiting neurologists and psychiatrists.

Wednesday, September 4, 1929.

9.30 a.m.—"The Educational Aspect of Stammering: A Plea for Research," by Dr. T. Garnet Leary, of Melbourne.

Demonstrations of pathological specimens, by Dr. Oliver Latham, of Sydney.

11 a.m.—With the Section of Ophthalmology: "The Value of Ocular Signs in Neurological Diagnosis," Dr. A. W. Campbell, of Sydney, Sir Henry Maudsley, of

Melbourne. Dr. D. D. Paton, of Perth, will take the chair. The meeting will be held in the practical physiology class room.

2 p.m.—Demonstration at the Glenfield Special School for Mentally Defective Children. The arrangements for this meeting will be carried out by the officers of the Department of Education of New South Wales.

Thursday, September 5, 1929.

9.30 a.m.—"Manic-depressive Psychoses," by Dr. Stuart Moore, of Dunedin.

10.15 a.m.—"Psychogenesis and Disease," by Dr. J. Williams, of Melbourne.

11 a.m.—With the Section of Medicine, the Section of Surgery and the Section of Orthopædics: "Trauma in Relation to Functional Nervous Disorders," Professor J. P. Lowson, of Brisbane, Dr. John Bostock, of Brisbane, Dr. W. M. Macdonald, of Dunedin, Dr. S. J. Minogue, of Callan Park, Dr. S. V. Sewell, of Melbourne. Dr. W. Ernest Jones will take the chair. The meeting will be held in the physiology lecture theatre.

2.30 p.m.—Clinical meeting at Broughton Hall Psychiatric Clinic, Wharf Road, Leichhardt.

Friday, September 6, 1929.

9.30 a.m.—"The Psychogalvanometer," by Professor W. S. Dawson, of Sydney.

10.15 a.m.—"Confusional Psychoses," by Dr. S. Evan Jones, of Broughton Hall.

11.45 a.m.—With the Section of Medicine: "Ventriculography and 'Lipiodol' in the Diagnosis of Diseases of the Central Nervous System," by Dr. J. F. Mackeddie, of Melbourne, followed by Dr. Lyle Buchanan, of Sydney. Dr. C. T. Ch. de Crespigny, of Adelaide, will take the chair. The meeting will be held in the physiology lecture theatre.

2 p.m.—Discussion on mental hygiene: "The Mental Hygiene Movement and Its Possibilities in Australia," by Dr. R. A. Noble, of Sydney.

"The Mental Hygiene Movement in New Zealand," by Dr. W. M. Macdonald, of Dunedin.

"The Value of Mental Hygiene to the Community," by Dr. John Bostock, of Brisbane, Dr. Henry F. Maudsley, of Melbourne.

Section of Obstetrics and Gynæcology.

Tuesday, September 3, 1929.

2.30 p.m.—Address by the President.

"Prolapsus Uteri: Morbid Anatomy," by Dr. Robert Fowler, of Melbourne.

"Prolapsus Uteri: Prevention and Treatment," by Dr. R. H. Morrison, of Melbourne.

Wednesday, September 4, 1929.

9.30 a.m.—"Ante Partum Hæmorrhage," by Dr. E. Brettingham-Moore, of Hobart.

"Post Partum Hæmorrhage," by Dr. J. A. Cameron, of Ipswich.

11 a.m.—"Pain and Other Reflexes in Labour," by Dr. Mary de Garis, of Geelong.

"A Note on the Control of Labour Pains by Morphine and Anæsthesia," by Dr. H. Leighton Kesteven, of Bullahdelah.

2.30 p.m.—Clinical demonstrations at the Royal Hospital for Women, Paddington, and at the Women's Hospital, Crown Street.

Thursday, September 5, 1929.

9.30 a.m.—"A Criticism of Modern Methods of Diagnosis and Treatment in Gynæcology," by Dr. A. N. McArthur, of Melbourne.

"Electro-thermic Myomectomy and Panhysterectomy," by Dr. Robert Fowler, of Melbourne.

Demonstration of a portable rubinization outfit, by Dr. R. Fowler, of Melbourne.

11 a.m.—With the Section of Pathology and Bacteriology: "Endometriomata," by Dr. Bernard Dawson, of Adelaide.

2.30 p.m.—"Maternal Mortality and Morbidity," by Professor H. Jellett, of Christchurch. It is hoped that Dame Janet Campbell will be able to open the discussion. Professor J. C. Windeyer, of Sydney, and Dr. E. S. Morris, of Sydney, will follow.

Friday, September 6, 1929.

9.30 a.m.—Demonstration at the Radium Clinic, by Dr. F. A. Maguire, of Sydney.

11 a.m.—With the Section of Pædiatrics and the Section of Preventive Medicine and Tropical Hygiene: "Natal and Neo-natal Mortality and Morbidity," by Professor R. Marshall Allan, of Melbourne, to be read by Dr. R. W. Chambers.

Section of Ophthalmology.

Tuesday, September 3, 1929.

2 p.m.—Address by the President, Dr. D. D. Paton, of Perth.

Wednesday, September 4, 1929.

9.15 a.m.—"Slit Lamp Microscopy of the Anterior Chamber," by Dr. R. Granville Waddy, of Sydney.

11 a.m.—With the Section of Neurology and Psychiatry: "The Value of Ocular Signs in Neurological Diagnosis," by Dr. E. A. Brearley, of Sydney.

Thursday, September 5, 1929.

9.15 a.m.—"Some Observations on the Prophylactic and Surgical Treatment of Early Cataract," by Dr. Guy Antill Pockley, of Sydney.

"Radiographs of the Anterior Half of the Globe without Bone Shadows," by Dr. H. F. Shorney, of Adelaide.

2 p.m.—Clinical meeting at the Royal Prince Alfred Hospital.

Friday, September 6, 1929.

9.15 a.m.—"The Treatment of Acute Glaucoma," by Dr. H. Coverdale, of

"Perimetry in the Early Diagnosis of Chronic Glaucoma," by Dr. E. C. Temple Smith, of Sydney.

2 p.m.—"The Treatment of Lachrymal Obstruction," by Dr. T. C. Ker, of Bendigo.

"Central Tarsorrhaphy," by Dr. J. C. Douglas, of Ballarat.

Section of Pædiatrics.

Tuesday, September 3, 1929.

2 p.m.—Address by the President, Dr. S. W. Ferguson, of Melbourne.

"Asthma and Allergic Conditions in Childhood," by Dr. C. Sutherland, of Melbourne.

Wednesday, September 4, 1929.

9.30 a.m.—"Human Serum in Poliomyelitis," by Dr. Jean Macnamara, of Melbourne.

11 a.m.—"Surgical Emergencies (Chest and Abdomen)," by Dr. Rupert Downes, of Melbourne.

12 noon.—"Electrocardiograms in Childhood," by Dr. H. L. Stokes, of Melbourne.

2 p.m.—Clinical meeting at the Royal Alexandra Hospital for Children, Camperdown.

Thursday, September 5, 1929.

9.30 a.m.—"Infant Feeding to the Age of Six Months," by Dr. H. Boyd Graham, of Melbourne.

11 a.m.—"Restoration of Breast Milk Feeding," by Dr. Guy Springthorpe, of Melbourne.

12 noon.—Visit to Tresillian Mothercraft Training School, Petersham.

2 p.m.—With the Section of Orthopædics and the Section of Radiology and Medical Electricity: "Bone Dystrophies," by Dr. R. B. Wade, of Sydney.

Friday, September 6, 1929.

9.30 a.m.—With the Section of Medicine, the Section of Oto-Rhino-Laryngology and the Section of Radiology and Medical Electricity: "Chronic Pulmonary Infections Secondary to Disease in the Upper Air Passages," by Dr. Douglas Galbraith, of Brunswick.

11 a.m.—With the Section of Obstetrics and Gynæcology and the Section of Preventive Medicine and Tropical Hygiene: "Natal and Neo-natal Mortality and Morbidity," by Dr. P. L. Hipsley, of Sydney.

2 p.m.—Clinical meeting at the Royal Alexandra Hospital for Children, Camperdown.

Section of Radiology and Medical Electricity.

Tuesday, September 3, 1929.

2 p.m.—Address by the President, Dr. H. M. Hewlitt, of Melbourne.

Wednesday, September 4, 1929.

9.30 a.m.—"Autocondensation in the Treatment of High Blood Pressure," by Dr. E. Payten Dark, of Katoomba.

11 a.m.—"Duodenal Diverticulum," by Dr. H. A. McCoy, of Adelaide.

Thursday, September 5, 1929.

11 a.m.—"Treatment by X rays, Radium *et cetera* of Growths of the Superficial Parts," by Dr. Herman Lawrence, of Melbourne.

2 p.m.—With the Section of Pædiatrics and the Section of Orthopædics: "Bone Dystrophies," by Dr. H. E. Sear, of Sydney.
Exhibition of films, by Dr. J. O'Sullivan, of Melbourne.

Friday, September 6, 1929.

9.30 a.m.—With the Section of Medicine, the Section of Oto-Rhino-Laryngology and the Section of Pædiatrics: "Chronic Pulmonary Infections in Relation to the Upper Respiratory Tract," by Dr. K. Stuart Cross, of Melbourne, Dr. J. Stanley Verco, of Adelaide, and Dr. E. Britten Jones, of Adelaide.

11 a.m.—With the Section of Surgery and the Section of Pathology and Bacteriology: "Bone Sarcoma," by Dr. J. Stanley Verco, of Adelaide.

Section of Surgery.

Tuesday, September 3, 1929.

2 p.m.—Address by the President, Professor Gordon Bell, of Dunedin, on diverticulitis.

Wednesday, September 4, 1929.

9.30 a.m.—"Malignant Disease of the Colon," by Mr. Victor Hurley, of Melbourne.

11 a.m.—With the Section of Orthopædics: "Compound Fractures of the Lower Extremity," by Mr. Fay Maclure, of Melbourne.

2 p.m.—"Follow-up Systems," by Dr. R. A. H. Fulton, of Dunedin.
"Prostatectomy Movies," by Dr. S. Harry Harris, of Sydney.
"Head Injuries," by Dr. V. M. Coppleson, of Sydney.
"Urogenital Tuberculosis," by Dr. R. Bridge, of Sydney.

Thursday, September 5, 1929.

9.30 a.m.—"Thyreoid Adenoma," by Dr. H. R. G. Poate, of Sydney.

11 a.m.—With the Section of Medicine, the Section of Neurology and Psychiatry and the Section of Orthopædics: "Trauma in Relation to Functional Nervous Disease," by Dr. H. S. Stacy, of Sydney.

2 p.m.—Clinical and operative work at hospitals; visit to the Radium Clinic at the Royal Prince Alfred Hospital.

Friday, September 6, 1929.

9.30 a.m.—Visit to the Urological Clinic at the Royal Prince Alfred Hospital.

11 a.m.—With the Section of Pathology and Bacteriology and the Section of Radiology and Medical Electricity: "Bone Sarcoma," by Sir Henry Newland, of Adelaide, Mr. Alan Newton, of Melbourne.

2 p.m.—Visits to metropolitan hospitals.

Congress Dinner.

The Congress dinner will be held on Wednesday, September 4, 1929. It is announced that those members who do not wish to be seated with the members attending a particular section, as previously arranged, may be seated in a group of general practitioners. If members will indicate their preference in their applications for dinner tickets, arrangements will be made accordingly.

Entertainments.

It will greatly assist the work of the Entertainments Committee if those members who are to be accompanied by ladies, will let the joint Honorary Secretaries know the names of the ladies, so that invitations and other communications may be suitably addressed.

Transactions of Congress.

The Transactions of the third session of the Australasian Medical Congress (British Medical Association), Sydney, 1929, will not appear in the form of supplements in THE MEDICAL JOURNAL OF AUSTRALIA as was the case in the first and second sessions of Congress, but will be published in one volume and will be distributed only to members of the Congress.

Correspondence.

THE SPLITTING OF FEES.

SIR: I realize that space in your journal is valuable, but I am constrained to reply to your comment on my letter published in the journal of June 29.

To use the words of your own definition of "fee-splitting," I have "handed a part of a fee received from a patient to another person for some service rendered in connexion with the patient."

For example I have handed a portion of the fee received to the anæsthetist for the administration of the anæsthetic or to an assistant or a consultant. You do describe this as an "illegal and objectionable practice," by your definition, and I consider my use of the words "arrogant and unwarranted" to be at least as "justified" as your "illegal and objectionable." Your definition in no way specifies any "secret commission."

I am fully aware that I do not practise splitting of fees in the "sense" to which exception *should be* (not always *is*) taken, but I do practise splitting of fees in the "non-sense" in which it is worded in the pledge under discussion.

I admit I am unfortunate (not singularly unfortunate) inasmuch as I cannot get a clear and not-ambiguous definition of fee-splitting. Surely the legal profession in Australia is not so feeble-minded as to be, if approached by the governing body of the College of Surgeons, unable to give a definition which could be included in the pledge, even though the best brains in the surgical profession be unequal to the task.

Yours, etc.,

"LUCIDEM."

Proceedings of the Australian Medical Boards.

VICTORIA.

THE undermentioned has been registered, under the provisions of Part I of the *Medical Act*, 1915, of Victoria, as a duly qualified medical practitioner:

Colvin, Henry David, M.B., Ch.B. (Edinburgh), 1901, F.R.C.S. (Edinburgh), 1908, M.D. (Edinburgh), 1910; 84, Powlett Street, East Melbourne.

QUEENSLAND.

THE undermentioned has been registered, under the provisions of *The Medical Act* of 1925, of Queensland, as a duly qualified medical practitioner:

Hinrichsen, William Henry, M.B., B.S., 1924 (Univ. Melbourne), Brisbane.

Restorations to the Register:

Cilento, Phyllis Dorothy, M.B., B.S. (Univ. Adelaide), Brisbane.

Deane, Maslen Mackenzie, M.B., B.S. 1926 (Univ. Melbourne), Brisbane.

Blackburn, John Herbert, L.R.C.P. and S. (Edinburgh), 1918, L.F.P. and S. (Glasgow), 1918; Boonah.

Books Received.

A DIABETIC MANUAL FOR THE MUTUAL USE OF DOCTOR AND PATIENT, by Elliott P. Joslin, M.D.; 1929. Philadelphia: Lea and Febiger; Sydney: Angus and Robertson, Limited. Post 8vo., pp. 248, with illustrations. Price: 10s. net.

SQUINT: ITS CAUSES, PATHOLOGY AND TREATMENT, by Claud Worth, F.R.C.S.; Sixth Edition; 1929. London: Baillière, Tindall and Cox. Demy 8vo., pp. 255, with illustrations. Price: 10s. 6d. net.

Diary for the Month.

AUG. 1.—South Australian Branch, B.M.A.: Council.
AUG. 2.—Queensland Branch, B.M.A.: Branch.
AUG. 6.—Tasmanian Branch, B.M.A.: Council.
AUG. 6.—Eye, Ear, Nose and Throat Section, South Australian Branch, B.M.A.
AUG. 7.—Victorian Branch, B.M.A.: Branch.
AUG. 7.—Western Australian Branch, B.M.A.: Council.
AUG. 8.—Victorian Branch, B.M.A.: Council.
AUG. 8.—New South Wales Branch, B.M.A.: Clinical Meeting.
AUG. 9.—Queensland Branch, B.M.A.: Council.
AUG. 9.—Eastern Suburbs Medical Association, New South Wales.

Medical Appointments.

The following appointments have been made in the "Mareeba" Babies' Hospital, South Australia: Responsible Honorary Advisory Medical Officer, Dr. Helen Mayo (B.M.A.); Honorary Medical Officers, Dr. F. H. Beare (B.M.A.); Dr. H. S. Covernton (B.M.A.), Dr. F. N. Le Messurier (B.M.A.); Honorary Consulting Aural Surgeon, Dr. R. H. Pulleine (B.M.A.); Honorary Pathologist, Dr. Annie Mocatta (B.M.A.); Honorary Assistant Pathologist, Dr. E. F. West (B.M.A.).

* * *

Dr. Jack Rupert Low Willis (B.M.A.) has been appointed an Honorary Medical Officer at the Mount Gambier Hospital, South Australia.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page XX.

AUSTIN HOSPITAL FOR CHRONIC DISEASES, HEIDELBERG, VICTORIA: Honorary Physician.

CHILDREN'S HOSPITAL, CARLTON, VICTORIA: Honorary Anæsthetists.

GIPPSLAND HOSPITAL, VICTORIA: Resident Medical Officer.

MENTAL DISEASES HOSPITAL, NEW NORFOLK, TASMANIA: Junior Medical Officer.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD: Honorary Clinical Assistants.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital. Toowoomba Friendly Societies Medical Institute.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.